

PHYSICS

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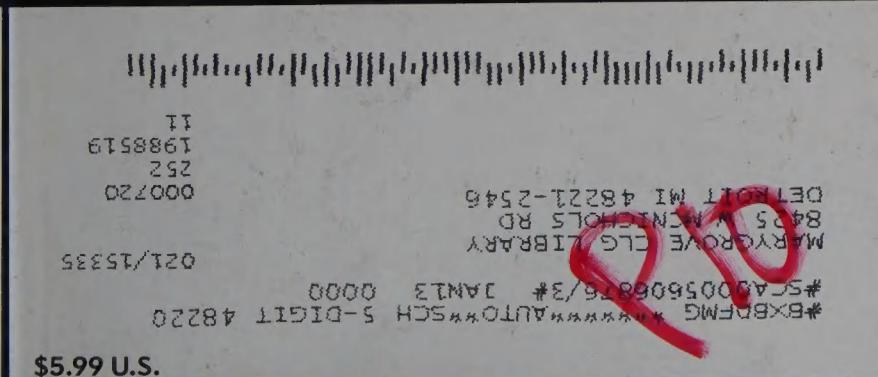
SCIENTIFIC AMERICAN

April 2012

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NORTHROP GRUMMAN



Recently discovered fossils from South Africa represent a species new to science, *Australopithecus sediba*, which could be the ancestor of our genus, *Homo*. The skull is part of a spectacularly well-preserved skeleton of a young male—one of several *A. sediba* individuals that died in an underground cave 1.977 million years ago outside what is now Johannesburg. Photograph by Brent Stirton, Getty Images.

SCIENTIFIC AMERICAN

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Sensational fossils from South Africa could revise long-held ideas about how we came to be human.
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Thousands of Physicists Descend on Boston

The American Physical Society's March meeting, the largest gathering of physicists in the world, covered everything from quantum optics to the physics of ponytails.

Go to www.ScientificAmerican.com/apr2012/aps

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Mariette DiChristina is editor in chief of *Scientific American*. Find her on Twitter @mdchristina



The Story Begins

HUMANS HAVE A SEEMINGLY PRIMAL NEED TO UNDERSTAND how we came to be the way we are today. Pieces of our ancient forebears generally are hard to come by, however. Scientists working to interpret our evolution often have had to make do with studying a fossil toe bone here or a jaw there. Now, in an amazing bounty, paleoanthropologist Lee Berger and his team have uncovered two well-preserved partial skeletons of *Australopithecus sediba* that date from nearly two million years ago at a site near Johannesburg, South Africa; the specimens include bones from every region of the body. The bones of at least four other individuals have also been found.

Already those first two *A. sediba* skeletons suggest we may have to revise a lot of what we thought was true about early human history. If Berger's interpretation of the bones is correct, some branches of our family tree may need to be redrawn. Senior editor Kate Wong visited the site with Berger to learn more. This month's cover story, "First of Our Kind," begins on page 30.

Turning to the state of our species today, we offer a special report on polio. Worldwide confirmed cases of the disease,

which once crippled hundreds of thousands, have dropped into the hundreds. But eliminating it remains challenging. Medical reporter Helen Branswell explains why in "Polio's Last Act," starting on page 60. As you'll learn in journalist William Swanson's "Birth of a Cold War Vaccine," starting on page 66, in the 1950s polio terrified the U.S. and U.S.S.R. almost as much as nuclear weapons. To test the oral vaccine, two scientists bridged their countries' differences. They have saved uncounted thousands from the misery of this ancient scourge. ■

SCIENCE IN ACTION

Last Call

Students ages 13 to 18, you still have time to enter the Google Science Fair: April 1 is the deadline. All entries are automatically considered for *Scientific American*'s Science in Action award. In addition to the \$50,000 prize, the winner will get a year of mentoring.

Science in Action is a new addition to the Google Science Fair for 2012. The winning project will be able to address a social, environmental or health issue to make a practical difference in the lives of a group or community and may possibly be scaled. To get inspired, watch a video about a Science in Action-style 2011 finalist, Harine Ravichandran, at www.ScientificAmerican.com/science-in-action. On that page, you can also find a list of our judges and mentors.

The Science in Action winner will be announced in June and will join Google Science Fair finalists at the company's Mountain View, Calif., campus for a special awards event on Monday, July 23. I'm a judge again this year. Best of luck to all entrants.

—M.D.

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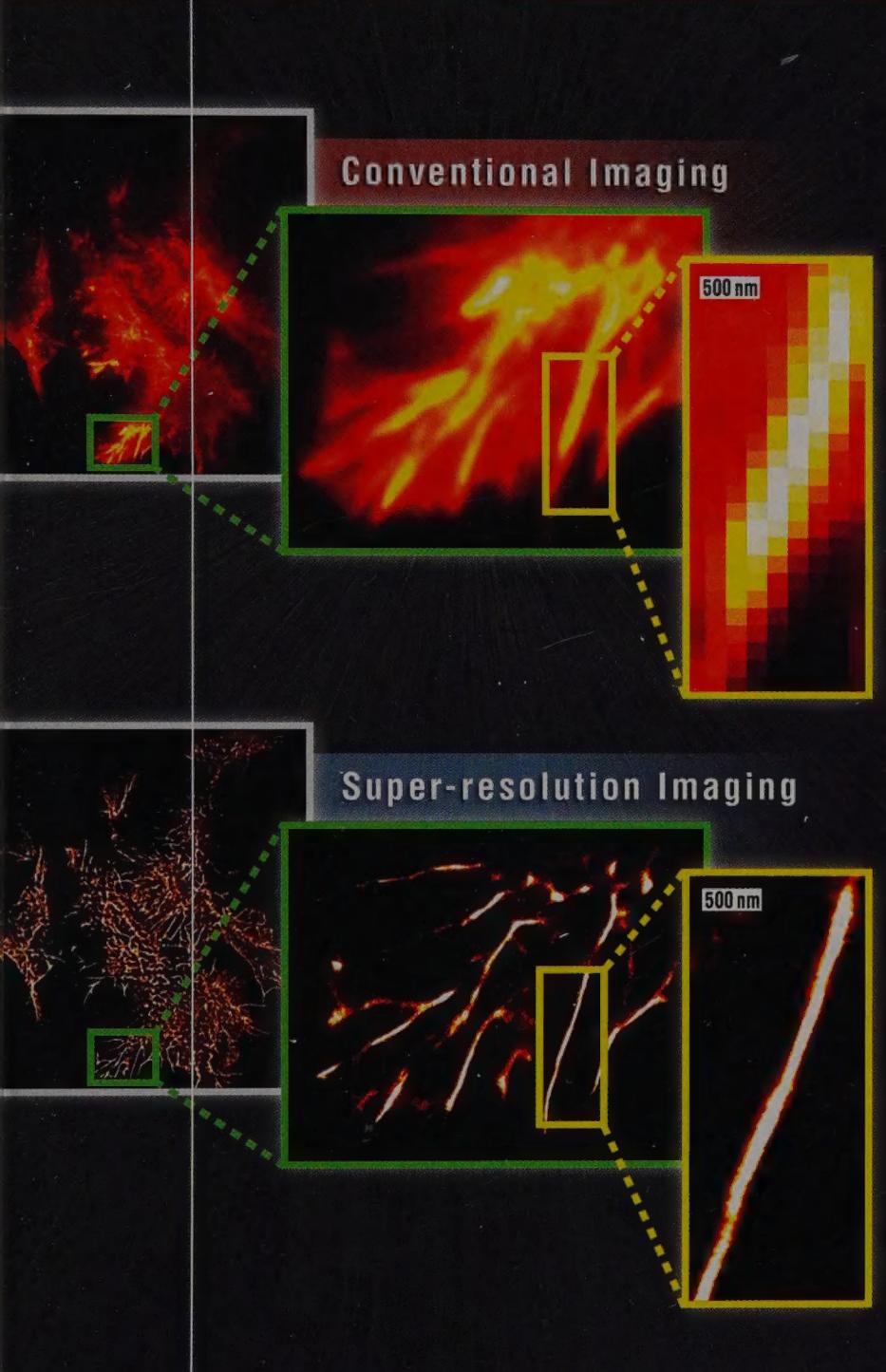
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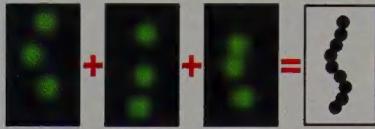
A stack of 2500 image frames was used to construct the upper, conventional microscopic image of actin bundles—which when magnified become indistinct. The lower image is constructed from 2500 frames from an ORCA-Flash camera. It shows dramatically improved resolution and clearly reveals individual actin bundles.*

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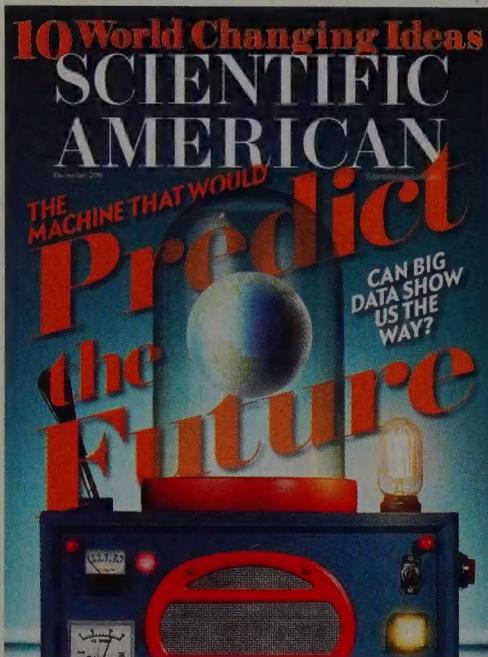
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December 2011

EPIGENETICS AND ANTIBIOTICS

"Hidden Switches in the Mind," by Eric J. Nestler, discusses epigenetic changes—alterations to how genes behave that do not affect the information they contain. Is it possible that such changes are at least partially responsible for bacteria becoming resistant to various drugs, given that the changes are passed on to daughter cells? If so, the changes would provide yet another way to overcome resistance to various drugs. Instead of looking for an entirely new antibiotic, it might be simpler to find a way to undo the epigenetic changes and restore the bacterial susceptibility to the drugs we already have.

TED GRINTHAL

Berkeley Heights, N.J.

EDITORS' NOTE: The author, not being a microbiologist, referred this question to Richard Losick, whose laboratory at Harvard University focuses on bacteria. Losick's reply follows:

Epigenetics does indeed contribute to antibiotic resistance in bacteria by giving rise to bacteria known as *persisters*. Indeed, epigenetic mechanisms were initially discovered in bacteria, although the mechanisms are quite different from the histone-based ones described in the article (bacteria do not have histones). *Persisters* are bacteria that survive antibiotic treatment without having acquired a resistance

"For the near future, we don't see large computing power successfully responding to the simple questions facing modern societies."

DAVID R. HARDY TORONTO

mutation. Instead they have reversibly entered a state in which they are less susceptible to killing by the antibiotic than other genetically identical cells in the population. Indeed, if we could devise drugs that blocked entry into the persister state, such drugs could contribute to the effectiveness of antibiotic therapy.

NOT OURS TO SEE?

Whereas David Weinberger's speculations about predictive abilities of big data-crunching models in "The Machine That Would Predict the Future" are intriguing, planners and social scientists aren't about to step aside just yet. As an example of "big data," IBM's Watson has impressive computing power when the question is clear, but important societal questions rarely are. For the near future, we don't see large computing power successfully responding to the simple questions facing modern societies with complex answers: For instance, how do you motivate Asian governments to take action on climate change? How do you reduce poverty? How do you get people out of their cars and onto public transit?

The challenge to prediction today is successfully integrating philosophy and the social and behavioral sciences with the physical sciences and engineering. Just witness the failure of climate scientists to advance the climate change agenda, resulting, in part, from social, behavioral and political scientists being left out of the conversation. With multidisciplinary cooperation as a starter, "big data" might be better equipped to predict the future.

DAVID R. HARDY

Toronto

I would suggest that there is an insurmountable hurdle to physicist Dirk Helbing's work, described by Weinberger, in trying to make a "computing system that would effectively serve as the world's crystal ball": the discrete architecture of the natural world. Helbing's background is apparently the modeling of highway traffic, which has a basic linear architecture. Road traffic acts like a hydraulic problem, where small particles can flow into one another continuously. My background is railroads, which couldn't behave more differently. On almost every level, their options and costs are effectively discrete. Railway costs are highly correlated, irregular, stepwise functions. They are dynamically unstable as they interact. That is, these costs are complexly unique lookup tables, not continuous equations, which means that highway and other linear models cannot be used rigorously (although people do try to use them).

So is the mathematical architecture of the world more like a road network or a rail network? If the latter, then it is mathematically impossible to predict the future. It would be a world ruled by discrete events, including black swan events. As a quote attributed to mathematician Benoît Mandelbrot put it, "Even though economics is a very old subject, it has not truly come to grips with the main difficulty, which is the inordinate practical importance of a few extreme events." God does, in fact, play with dice in the natural world.

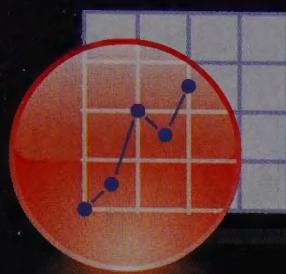
TOM ERICKSON
Wallingford, Pa.

COFFEE AND MINUETS

Charles Q. Choi's reportage of Rouslan Krechetnikov's study on the physics of keeping coffee from spilling, as reported in "Fluid Dynamics in a Cup" [Advances], is interesting. But Choi should go, at peak dinnertime, to a restaurant known for very good service. Watch the wait staff move quickly across the floor. When they are carrying liquids, the rule is long stride, short stride, long stride, short stride.

You can walk briskly and not spill things if you're careful to break up your rhythm as you move. I learned that the first day on the job as a waiter in the 1960s.

ROD RODUIN
via e-mail



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Keith J. Stevenson

Journal of American Chemical Society, March 2011

In a nutshell, Origin, the base version, and OriginPro, with extended functionality, provide point-and-click control over every element of a plot. Additionally, users can create multiple types of richly formatted plots, perform data analysis and then embed both graphs and results into dynamically updated report templates for efficient re-use of effort.

Vince Adams

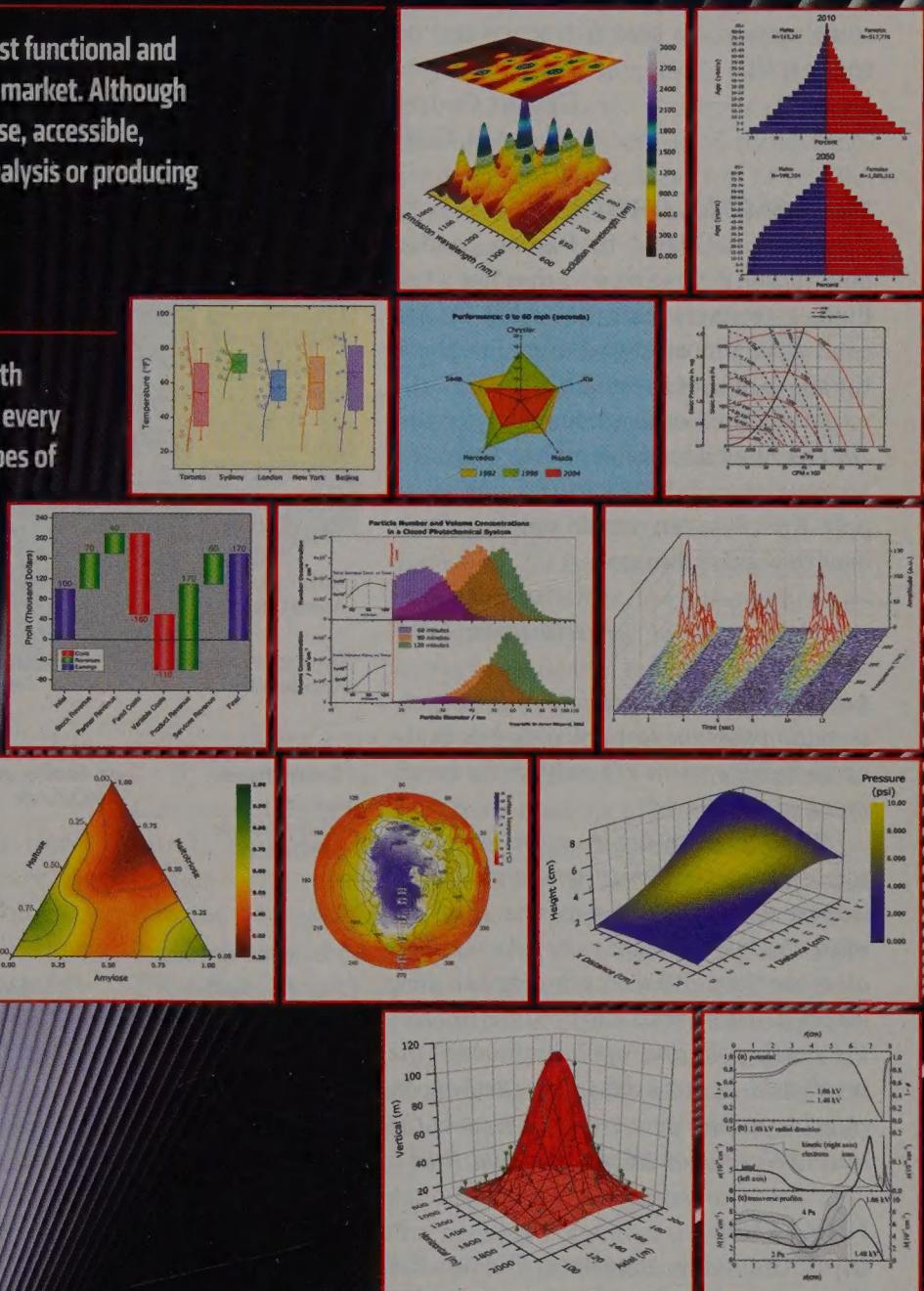
Desktop Engineering, July 2011

I have used Origin for years. It gives me the ability to control every aspect of the graph I am creating. This flexibility combined with its statistical tools have made Origin an indispensable part of my daily work.

Scott Jackson, Ph.D.

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I believe a solution for spilling coffee was already discovered 35 years ago on the University of California, Berkeley, campus. At the time, the student union lacked lids for coffee, and inevitably some of the precious brew would slosh out before I reached my 8 a.m. class. Realizing the problem was a buildup of vibrations until a large "beat" frequency caused the liquid to spill, I tried breaking them up by randomly moving the cup side to side and fore and aft as I walked. Eureka! No constant motion, no beat frequency and no coffee spilt.

GEORGE COOPER
Alameda, Calif.

IMPOSSIBLE ORBIT?

"This Way to Mars," by Damon Landau and Nathan J. Strange, says that "for a test flight, astronauts steer the vehicle into an orbit that almost always remains above the south pole of the moon."

As a retired orbit mechanic, I see this statement as impossible unless there is a Lagrangian point above the lunar south pole. A satellite can remain stationary only over the body's equator.

RICHARD BOBROW
Westminster, Calif.

LANDAU REPLIES: I appreciate Bobrow's observation that the stationary points in the earth-moon system are only in the earth-moon plane.

When writing, however, we were sure to put in "weasel words" where we did not want to open up a can of worms. Here "almost always" is meant to be taken as "not all of the time." We were alluding to a very elliptical orbit with a low perilune "above" the north pole and apolune "above" the south pole. In our orbit in the earth-moon rotating frame, we are within view of the south polar region 96 percent of the time.

ERRATUM

In the interview "Speaking Out on the 'Quiet Crisis,'" by Brendan Borrell, Shirley Ann Jackson, president of the Rensselaer Polytechnic Institute, is referred to incorrectly as the first African-American woman to receive a Ph.D. In 1973 she was the first African-American woman to receive a Ph.D. from the Massachusetts Institute of Technology.

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Who Owns the Past?

The federal government should fix or drop new regulations that throttle scientific study of America's heritage

A rare set of nearly 10,000-year-old human bones (right) found in 1976 on a sea-side bluff in La Jolla, Calif., may soon be removed from the custody of the University of California, San Diego, and turned over to the local Kumeyaay Nation tribes. The Kumeyaay have long sought control over the bones, which they contend are the remains of their ancestors. In accordance with new federal regulations, the university has initiated the legal process to transfer the remains to the Kumeyaay in the absence of other claimants. The Kumeyaay have said they may rebury the bones. Being some of the oldest human skeletal remains in North America, the bones could help scientists piece together the peopling of the New World. The excellent preservation of the specimens hints that they might contain DNA suitable for analysis with techniques geneticists have recently developed—the results of which could yield crucial insights into where early Americans came from. Such studies may never come to pass.

Some might consider a loss of knowledge an acceptable trade-off to right the historic wrongs that the Kumeyaay and other Native peoples have suffered. Archaeologists and anthropologists of yore treated Native Americans disgracefully, looting their graves and using the remains to argue for the intellectual inferiority of Native Americans to peoples of Caucasian descent. But what makes this case disturbing is that the Kumeyaay claim is based on folklore. The physical evidence indicates that the La Jolla bones are not affiliated with any modern tribe, including the Kumeyaay, who moved into the area only within the past few thousand years. The new federal regulations are blind to this evidence. In effect, they privilege faith over fact.

The original intention of the Native American Graves Protection and Repatriation Act (NAGPRA), passed in 1990, was to facilitate the return of Native American bones and sacred objects to descendants and culturally affiliated groups. NAGPRA sought to balance the rights of Native Americans to reclaim ancestral remains with the right of society as a whole to learn about our collective past. By and large, the law was succeeding. In recent years scientists and representatives of Native peoples have been working together to everyone's gain. For example, archaeologist Alston



Thoms of Texas A&M University has been consulting with Native Americans about their cooking techniques, to gain insights into the subsistence strategies of people who lived on the South Texas plains thousands of years ago. Members of the Tap Pilam Coahuiltecan Nation—who consider themselves the descendants of those ancient Texans—have, in turn, been learning about ancestral foods and incorporating them into their diet to counter the high rate of diabetes in their population.

Many Native Americans do not object to studies *per se* but to analyses that destroy remains. Respecting this concern, anthropologist Ventura Pérez of the University of Massachusetts Amherst, who studies violence, has developed techniques for making high-quality replicas of cut marks on bone that leave the skeletal material intact and allow it to be repatriated,

while creating a permanent record for future scholars.

To be sure, not all was well. Many tribes worried that museums were stalling on identifying remains to avoid having to return them. In May 2010 the U.S. Department of the Interior responded with regulations that allow tribes to claim even those remains whose affiliation cannot be established scientifically, as long as they were found on or near the tribes' aboriginal lands. These rules nudge museums to get on with evaluating their collections, but they have too broad a brush. They upset the balance that NAGPRA had achieved and foster antagonism, not just between tribes and scientists but also among tribes with conflicting claims. The La Jolla case is just one example. Thousands of remains could be made inaccessible to researchers. In our view, the new regulations should be repealed or, at least, revised to distinguish different classes of unidentified remains.

The colonization of the New World was a watershed in the odyssey that carried *Homo sapiens* from its African birthplace to the entire globe. The stories of the trailblazers who accomplished that feat deserve to be told. Their remains are the shared patrimony of all Americans and, indeed, all peoples everywhere. ■

SCIENTIFIC AMERICAN ONLINE

Comment on this article at ScientificAmerican.com/apr2012

Stuart Firestein is a professor and chair of biological sciences at Columbia University. He is author of *Ignorance: How It Drives Science*, which Oxford University Press is releasing this month.



What Science Wants to Know

An impenetrable mountain of facts can obscure the deeper questions

Most scholars agree that Isaac Newton, while formulating the laws of force and gravity and inventing the calculus in the late 1600s, probably knew all the science there was to know at the time. In the ensuing 350 years an estimated 50 million research papers and innumerable books have been published in the natural sciences and mathematics. The modern high school student probably now possesses more scientific knowledge than Newton did, yet science to many people seems to be an impenetrable mountain of facts.

One way scientists have tried to cope with this mountain is by becoming more and more specialized, with limited success. As a biologist, I wouldn't expect to get past the first two sentences of a physics paper. Even papers in immunology or cell biology mystify me—and so do some papers in my own field, neurobiology. Every day my expertise seems to get narrower. So scientists have had to fall back on another strategy for coping with the mountain of information: we largely ignore it.

That shouldn't come as a surprise. Sure, you have to know a lot to be a scientist, but knowing a lot is not what *makes* a scientist. What makes a scientist is ignorance. This may sound ridiculous, but for scientists the facts are just a starting place. In science, every new discovery raises 10 new questions, as playwright George Bernard Shaw sardonically declared in a dinner toast to Albert Einstein.

By this calculus, ignorance will always grow faster than knowledge. Scientists and laypeople alike would agree that for all we have come to know, there is far more we don't know. More important, everyday there is far more we *know* we don't know. One crucial outcome of scientific knowledge is to generate new and better ways of being ignorant: not the kind of ignorance that is associated with a lack of curiosity or education but rather a cultivated, high-quality ignorance. This gets to the essence of what scientists do: they make distinctions between qualities of ignorance. They do it in grant proposals and over beers at meetings. As James Clerk Maxwell, probably the greatest physicist between Newton and Einstein, said, "Thoroughly conscious ignorance ... is a prelude to every real advance in knowledge."

This perspective on science—that it is about the questions more than the answers—should come as something of a relief. It



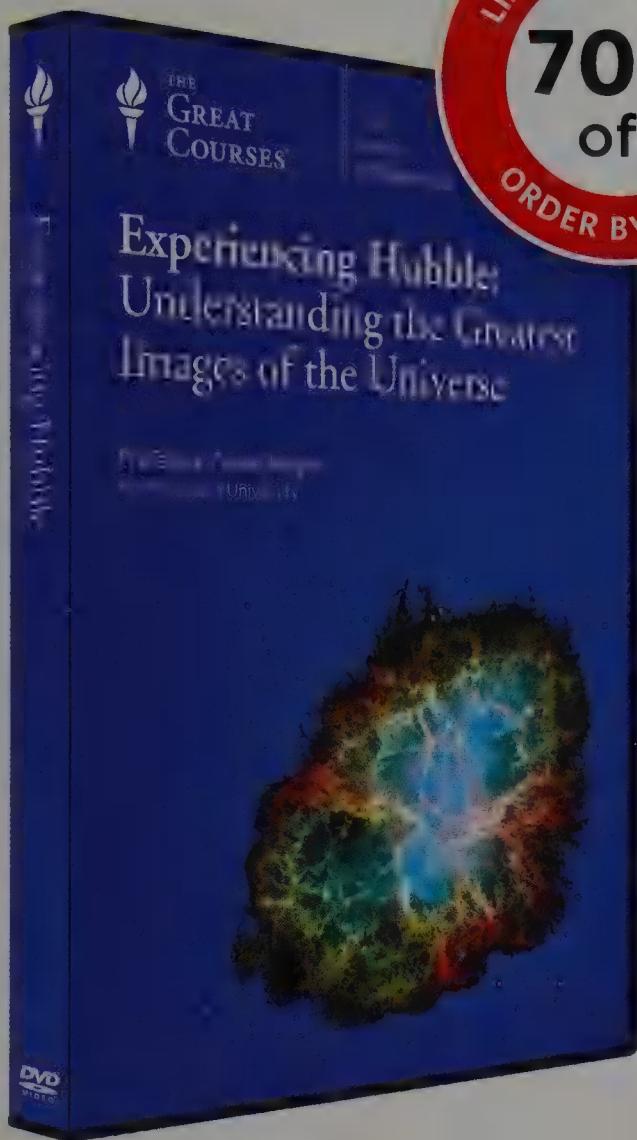
makes science less threatening and far more friendly and, in fact, fun. Science becomes a series of elegant puzzles and puzzles within puzzles—and who doesn't like puzzles? Questions are also more accessible and often more interesting than answers; answers tend to be the end of the process, whereas questions have you in the thick of things. I can't grasp much of immunology even though I have a fancy Ph.D., but the wonderful thing is that most immunologists can't either—no one knows everything anymore. I can, however, understand the questions that drive immunology. And although I don't pretend to understand much about quantum physics, I can appreciate how the questions in that field arise and why they are so fundamental. Emphasizing ignorance is inclusive; it makes everyone feel more equal in the same way the infinity of space pares everyone down to size.

Of late this side of science has taken a backseat in the public mind to what I call the accumulation view of science—that it is a pile of facts way too big for us to ever hope to conquer. But if scientists would talk about the questions rather than bore your eyes out of their sockets with reams of jargon, and if the media reported not only on new discoveries but the questions they answered and the new puzzles they created, and if educators stopped trafficking in facts that are already available on Wikipedia—then we might find a public once again engaged in this great adventure that has been going on for the past 15 generations.

So if you meet a scientist, don't ask her what she knows, ask her what she *wants* to know. It's a much better conversation—for both of you. ■

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NEUROSCIENCE

The Mind Recovery Act

Why Obama's "War on Alzheimer's" may pay off

Government declarations of war on drugs or disease often end in losing battles. That is why some neuroscientists have greeted the Obama administration's goal of preventing or treating Alzheimer's by 2025 with skepticism. "Setting target dates for any research program always carries the danger of falsely raising expectations," says Kenneth S. Kosik, a professor of neuroscience at the University of California, Santa Barbara. "Research does not function like an assembly line in which we can project outcomes." President Barack Obama signed the National Alzheimer's Project Act into law more than a year ago, and the White House handed in a draft of the plan to Secretary of Health and Human Services Kathleen Sebelius in February. The proposal includes \$50 million in new research funding for this year.

The 2025 deadline is not as unrealistic as it might seem. In war, anticipating the enemy's next move is half the battle, and some of the most meaningful advances in Alzheimer's research in recent years have to do with reconnaissance. Studies have shown that magnetic resonance imaging, positron-emission tomography and spinal taps—and newer methods now in the lab—can detect the effects of the buildup of aberrant proteins characteristic of Alzheimer's some 10 to 15 years before the first symptoms appear. They may be able to go back further, identifying a persistent inflammatory response deep within the brain or capturing the period when mitochondria, the cellular powerhouses, begin spewing toxins as early as middle age. These are normal accompaniments of aging in all of us. For some, however, these changes interact with bad genes or other unidentified risk factors to initiate the tortuously slow process that ends with dementia.

Leading research groups are already calculating what they can do with this molecular intelligence report. In February a group of researchers from Case Western Reserve University reported online in *Science* that a cancer drug with relatively benign side effects was able to rapidly clear from the brains of mice toxic amyloid-beta protein fragments that accompany Alzheimer's. The compound is headed to human trials and, if it proves its mettle, could perhaps be a prelude to a statinlike drug for dementia.

For now biomarkers, as researchers call them, offer the best hope for establishing a path toward staving off cognitive decline—and for meeting the Obama administration's ambitious goal.



—Gary Stix

MRI SCAN shows damage typical of Alzheimer's disease.

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ENGINEERING

Scanning for E.T.'s Calls

A giant telescope will soon begin its search for the first stars and galaxies

More than 44,000 radio antennas will soon link over the Internet to create one of the most ambitious radio telescopes ever built. Its job will be to scan largely unexplored radio frequencies, hunting for the first stars and galaxies and, potentially, signals of extraterrestrial intelligence.

The array is designed to monitor low-frequency radio

waves. One key source of these emissions are extraordinarily feeble signals from the cold hydrogen gas that dominated the cosmos during the so-called Dark Ages of the universe. As stars eventually flared into being, they would have left scars on this hydrogen, and by analyzing how the radio signals from this gas altered over time, sci-

entists can learn much about how the first galaxies came to be.

The Low Frequency Array (LOFAR) will consist of banks of antennas in 48 stations in the Netherlands, Germany, France, Sweden and the U.K., all connected via fiber-optic cables. A supercomputer will combine signals from these stations, transforming the

array into what may be the most complex and versatile radio telescope ever attempted, says Heino Falcke, chair of the board for the International LOFAR Telescope.

The array will be finished by the middle of this year and will have the capacity to sweep the entire northern sky in 45 days. All told, it will have a maximum resolution equivalent to a telescope 620 miles (1,000 kilometers) in diameter. In addition, the design is expandable, meaning that researchers can always add stations later, says Michael Wise of ASTRON, the Netherlands Institute for Radio Astronomy.

In addition, LOFAR is very fast, capable of measuring events only five billionths of a second long. Moreover, the fact that LOFAR is essentially many different radio telescopes knit together means it can run, say, three different science projects simultaneously, Wise says.

In the next few years, the array will also scan for artificial radio emissions as part of the search for extraterrestrial intelligence (SETI) at lower, neglected frequencies than past SETI missions.

—Charles Q. Choi
and Space.com

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BY THE NUMBERS

\$54.5 MILLION

Amount Mitt Romney has raised for his presidential campaign as of Jan. 31, including super PAC funding

\$44.3 MILLION

Amount Barack Obama has raised for his reelection as of Jan. 31, including super PAC funding

Amount the National Cancer Institute spent in 2010 on esophageal research

\$30.5 MILLION

Amount the National Science Foundation requested in 2011 for the Gemini Observatory, among the most powerful telescopes in the world

\$19.6 MILLION

\$18 MILLION

What the NSF spent in 2010 to fund the Large Hadron Collider

\$14 MILLION

Estimated amount the National Institutes of Health spent in 2011 funding gene therapy clinical trials

Hopeful Vision

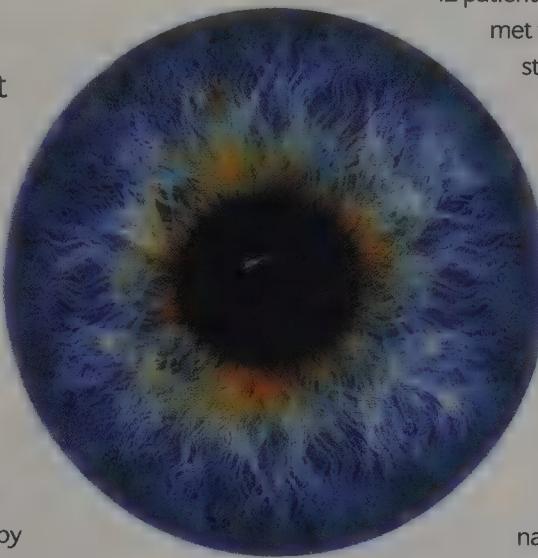
Gene therapy restores human sight

After several years of setbacks, gene therapy is once again yielding promising results. One area in which it is proving its potential is in restoring vision to patients who have been losing it since birth.

Between 2008 and 2011 Jean Bennett, a neuroscientist at the University of Pennsylvania, and her colleagues used gene therapy to treat blindness in 12 adults and children with Leber's congenital amaurosis (LCA). LCA is a rare inherited eye disease that destroys vision by killing photoreceptors—light-sensitive cells in the retina at the back of the eye. Typically afflicted children start life with poor vision, which worsens as more and more photoreceptors die.

The treatment grew out of the understanding that people with the disorder become blind because of genetic mutations in retinal cells. Once such mutation prevents the production of an enzyme needed to break down retinol, a form of vitamin A, into a substance that photoreceptors need to detect light and send signals to the brain.

In their original study, Bennett and her colleagues treated each of the



12 patients in one eye; six improved so much they no longer met the criteria for legal blindness. In a subsequent study published in February in *Science Translational Medicine*, the researchers injected functional genes into the previously untreated eye in three of the women from the first group and followed them for six months. The women's vision in their previously untreated eye improved as soon as two weeks after the operation: they could avoid obstacles in dim light, read large print and recognize faces.

Bennett showed that not only were the women's eyes much more sensitive to light, their brains were much more responsive to optical input as well. Functional magnetic resonance imaging showed regions of their visual cortex lighting up that had been offline before gene therapy began. Surprisingly, the second round of gene therapy

further strengthened the brain's response to the initially treated eye, as well as the newly treated one, perhaps "because the two eyes act in concert, and some aspects of vision rely on binocular vision," she says.

Bennett thinks the therapy will work even better in younger patients who have not lost as many photoreceptors. The results "really bode well" for restoring meaningful vision to people with LCA and other forms of inherited blindness, Bennett says.

—Ferris Jabr

PROMOTION

WHAT'S THE FUTURE OF ENERGY? HERE'S WHAT YOU'RE TELLING US.

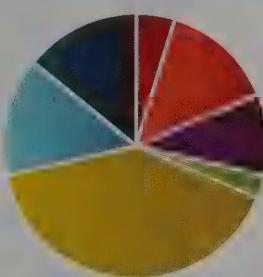
Shell is posing provocative questions to start a conversation about this important topic.

The Energy for the Future Poll measures global and regional opinions on where to place our energy and transportation priorities.



Which area of the energy industry will advance the most in the next 10 years?

40% of the worldwide respondents thought that the solar industry offers the most potential technological progress. The wind power, biofuels and nuclear industries each earned about 14% of the votes. In Asia and Australia, respondents ranked nuclear as a clear second, while respondents from Africa see biofuels as the #2 most-promising.



What policy option do you think is the most effective as a solution to the energy challenge?

32% of global respondents saw mandating alternative energy as the most effective policy, while subsidizing renewable energy (18%) and carbon taxes (14%) were also strongly supported. In the lively online discussion, a number of respondents pointed out that controlling energy consumption and minimizing distribution waste are important steps we can take today.



Look for the results of the Shell Energy for the Future Poll on scientificamerican.com/sponsored/energyforthefuture



Picky Eaters Club

Fungi that orchids need to grow are just as finicky as the exotic flowers themselves

In The Orchid Thief, writer Susan Orlean describes the cultlike devotion that these exotic-looking flowers inspire among plant collectors. One reason, in addition to their beauty, that orchids are so prized is that they are fragile: although they grow in every U.S. state and on every continent except Antarctica, many are endangered, and the flowers are exceedingly sensitive to environmental changes. Native orchids' dustlike seeds will grow only if nourished by certain groups of root fungi, known as mycorrhizal fungi.

Little is known about these organisms—so little that many have not been named. They grow into the roots of orchids, which digest the fungi to obtain needed nutrients. Recently a four-year study has shed new light on where mycorrhizal fungi grow and under what conditions they stimulate orchids to germinate. The results, published online January 24 in *Molecular Ecology*, will help ecologists preserve rare orchid varieties.

The team of researchers, led by ecologist Melissa McCormick of the Smithsonian Environmental Research Center in Edgewater, Md., planted and tracked three U.S. orchid species—all present in the East and endangered somewhere in the country—in six study sites:

three in younger forests, which were 50 to 70 years old, and three in older forests, which were 120 to 150 years old. Investigators covered each plot with leaf litter, decomposing wood or nothing and provided half the plots with the specific fungi known to promote growth in each orchid.

The researchers also identified the existing fungi in each forest. Because the organisms have no fruiting structures, they can be tough to detect, so the team pioneered the use of testing for DNA in the soil to identify

where and how much fungus was present. Older forests, McCormick and her colleagues found, had about five to 12 times more orchid-friendly fungi than younger forests, and the fungi in older forests were more diverse.

Each orchid had different requirements to grow. For *Goodyera pubescens* (a stalk of its small white flowers is pictured), only older forests held enough fungus for it to flourish. Adding the fungus to younger forests alone or in combination with decomposing wood did not make *Goodyera* seeds germinate.

The host fungus of *Tipularia discolor*, which has many small mauve-purple flowers with yellowish centers, was widespread in young and old forests alike but could support germination only on decomposing wood. The host fungus for *Liparis liliifolia* wasn't common in the wild, but the orchid would germinate if the fungus was added.

Orchid conservation plans generally do not account for orchid fungi abundance or requirements, simply because the techniques and knowledge to identify the fungi haven't been in place. Says McCormick, "We're hoping others can apply these techniques to figure out what environmental conditions affect the fungi."

—Carrie Madren



PATENT WATCH

Proprioceptive feedback system: When Hilary Mass was eight years old, her family added a new member who would change her life forever. Her little brother was born with special needs, and as the oldest child Mass found herself in a new position. "I was always the helper," she says, "so I always knew this was what I was going to do."

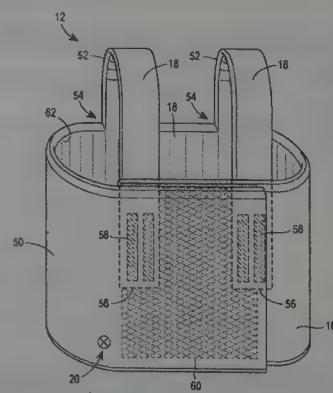
For the past 30 years she has worked with special-needs kids. As Mass spent time with her charges, she noticed something that she thought could be better: many children with special needs have what specialists call sensory diets—activity plans that ensure their unique sensory needs are met. Some autistic children, for example, like to be hugged or have their shoulders pressed on—they find such touching calming, and it allows them better control over their anxiety and hyperactivity.

To achieve that sensation, many of them wear a weighted vest with sandbags to provide pressure on their shoulders and elsewhere. But Mass noticed that the children did not like wearing this bulky vest even if they liked the result. "My students don't choose to put it on," she says. "You have to talk them into it or force them to wear it."

Seven years ago Mass began developing her own system. What she came up with—patent No. 8,095,994, which she is calling Big Hug—is a suit that children could wear just like an article of clothing. Mass's design does not rely on sandbags or weights like the standard vests but instead inflates with air to create even and customizable pressure on the body.

She has tested it only on her own students and children she knows, but so far it has been a success. "The students really seem to like it," she says. "They ask for it." Mass is now trying to put Big Hug on the market. "My motivation was to help families to make things a little easier when they're trying to get through the day," she says.

—Rose Eveleth





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Fast Talkers

Some languages sound faster than others, but most convey information at the same rate

“Speakers of some languages seem to rattle away at high speed like machine-guns, while other languages sound rather slow and plodding,” wrote linguist Peter Roach in 1998. A few months ago researchers systematically quantified Roach’s observation and offered a surprising explanation. Last year, in an issue of the journal *Language*, François Pellegrino and his colleagues at the University of Lyon in France published their analysis of the speech of 59 people reading the same 20 texts aloud in seven languages. They found Japanese and Spanish, often described as “fast languages,” clocked the greatest number of syllables per second. The “slowest” language in the set was Mandarin,

followed closely by German.

But the story does not end there. The researchers also calculated the information density for the syllables of each language by comparing them with an eighth language, Vietnamese, which served as an arbitrary reference. They found that an average Spanish syllable conveys only a small quantity of information, contributing just a fragment to the overall meaning of a sentence. In contrast, an individual Mandarin syllable contains a much larger quantity of information, possibly because Mandarin syllables include tones. The upshot is that Spanish and Mandarin actually convey information to listeners at about the same rate. The correlation between

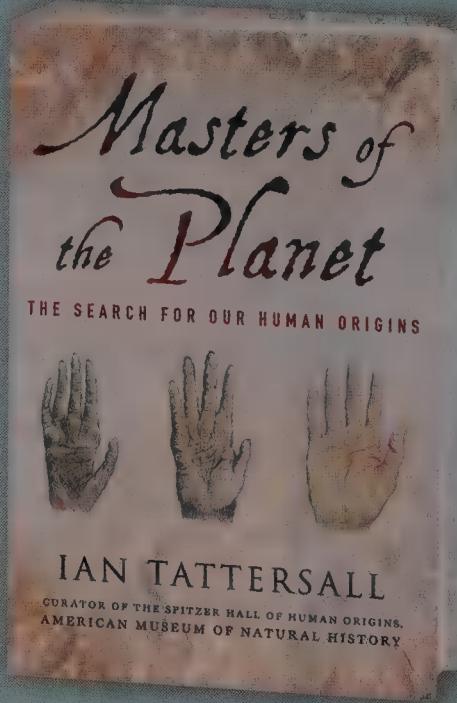


speech rate and information density held for five out of seven of the languages studied, and the researchers conjectured that, despite the diversity of languages in the world, over time they all deliver a constant rate of information, possibly tuned to the human perceptual system.

The results of these studies could change the way we think about the diversity of the world’s languages. In the 1950s linguist Noam Chomsky proposed the idea of universal grammar, which suggests that all languages, their apparent differences notwithstanding, possess a common set of abstract structures. This hypothesis galvanized the field of linguistics, but truly common structures proved tough to find. The current research suggests that languages can and do use a wide variety of structures, as long as they deliver information to listeners at a relatively constant rate. Thought of in this way, universal grammar is no longer an abstract notion but a linchpin of human communication that ensures a steady flow of information from speaker to listener. —Anne Pycha

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English	6.19 (± 0.16)
German	5.97 (± 0.19)
Vietnamese	5.22 (± 0.08)
Mandarin	5.18 (± 0.15)

Blue Bacteria in Bloom

The proliferation of cyanobacteria in oceans may accelerate warming

On their own, cyanobacteria are tiny photosynthetic organisms floating in the sea. But when they join forces, linking together into chains and then mats by the millions, they can become a threat. Before long, the bacteria change the color of the sea's surface and even soften the wind-tossed chop. One study of cyanobacteria, also known as blue-green algae, although they are not algae, predicted that rising sea temperatures could help the already widespread creatures expand their territory by more than 10 percent. Now researchers are asking whether mats of cyanobacteria might themselves affect local sea temperatures, thus creating a powerful feedback loop.

Cyanobacteria are ubiquitous. They spew enough oxygen into the atmosphere to dictate the current mix of gases we breathe. They also compete—with great success—for nutrients such as nitrogen and phosphorus. When cyanobacteria bloom, it is often at the cost of neighboring species such as fish or other phytoplankton. So if cyanobacteria are shaping the temperature of their growing patch of the ocean to favor themselves over cold-

water critters, researchers want to know how they are doing it and what to expect next, says climate scientist Sebastian Sonntag of the University of Hamburg in Germany.

Sonntag and his colleagues have adapted a computer model that describes the mixing of layers of seawater to take into account two kinds of changes produced by the cyanobacterium *Trichodesmium*: more light absorption and less choppy waves. The updated model predicted sea-surface warming of up to two degrees Celsius because of light absorption. The wave damping appeared to affect local temperatures by about one degree C.

This may be the first such study of algal blooms in the ocean, says aquatic microbiologist Jef Huisman of the University of Amsterdam, who has studied light absorption by cyanobacteria in lakes. Both Sonntag and Huisman say they would like to ask oceanographers to measure seawater temperature where cyanobacteria grow and in nearby empty areas to test the new model's predictions and to improve future versions.

—Lucas Laursen



COURTESY OF ELIZABETH C. SARGENT/University of Southampton and National Oceanography Center, Southampton



FIELD NOTES

Coffee Mystery

An entomologist describes his efforts to stop Rwanda's coffee from tasting like potatoes

Potato taste is a category of coffee taste, and the name is as close as professional coffee tasters can come to describing it. The term winds up categorizing that batch of coffee as undesirable.

Rwanda started having problems with potato taste about four years ago, after they started concentrating on specialty coffee. The difference between specialty coffee and regular coffee is like night and day. Specialty coffee re-

quires a lot more work, but its price is relatively stable compared with ordinary coffee. That means that coffee represents a whopping percentage of their income. We're talking millions of dollars that could be lost to potato taste.

I recently joined a small team to give presentations in Rwanda. They set up a tasting for us in a Starbucks, where they have a micro roaster and micro grinder on-site. The taste is very subtle, of course, but to me it had a musty sort of smell that reminded me of old cardboard paper.

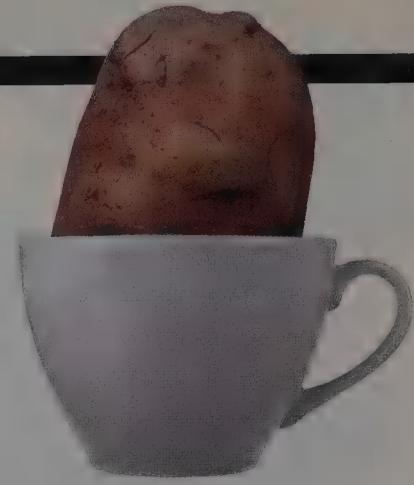
We suspect that a microbe is involved, and it might be associated with a group of stink-bugs called antestia. When potato taste comes from a batch, and it can be traced back to an origin, the district is usually infested with antestia. So

there's a loose correlation with the insect. And the antestia bug by itself, regardless of this taste, damages 35 percent of the coffee yield.

My recommendation to Rwanda is to try to reproduce the system. You've got the insect, you've got the coffee bean, and you put those in a model system and turn the crank. Let's say you come up with a really bad potato taste, then you can go backward and see where the microbe comes into the equation.

In the meantime, Rwanda can try to deal with the antestia bug because it damages coffee yield anyway. The bug lives on abandoned coffee plantations and in the mulch under banana trees, so if you do some sanitation work to get rid of it, that could help in two ways.

The trouble with the potato



PROFILE

NAME

Thomas Miller

TITLE

Professor of entomology

LOCATION

University of California, Riverside

taste defect is you can't see it. It's not really clear what steps you need to take to prevent it from happening. It is a great big mystery. You've got yourself a nice 20 years of work before you figure out what's causing it. —As told to Rose Eveleth

EDUCATION

Textbooks Come Alive

Next-generation science e-books may help keep young people engaged

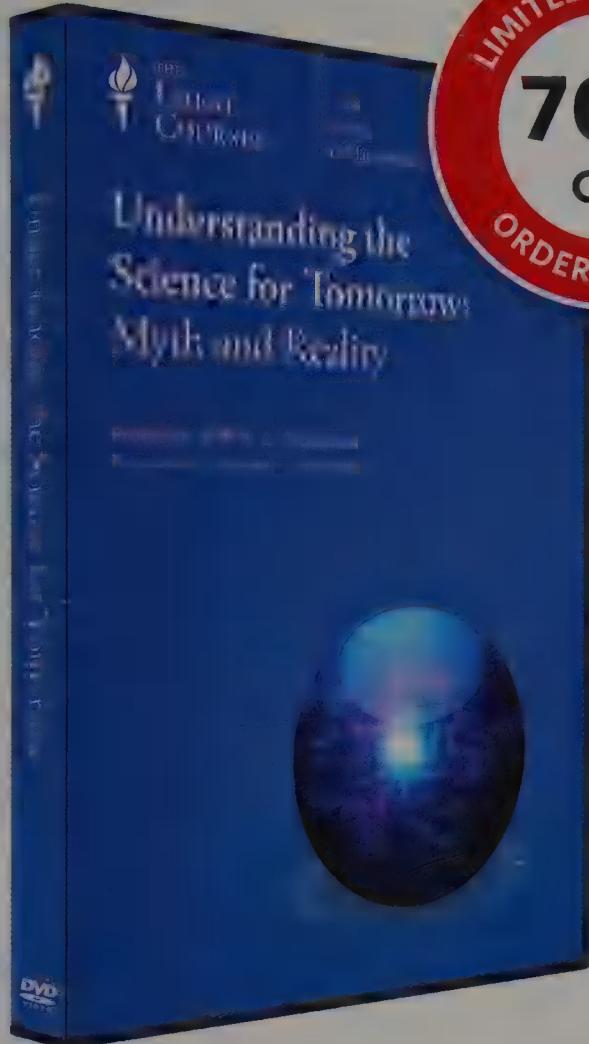
Science can advance quickly, rendering existing textbooks obsolete. Now **new** digital textbooks **are emerging** intended to better engage students and keep them up-to-date on the latest research. These e-books will cost (and weigh) less than the average printed tome. In January, Apple announced its iBooks 2 textbook platform for the iPad, and publishers, including McGraw-Hill, Pearson, and Houghton Mifflin Harcourt, have signed on to create content for it. In February, Nature Publishing Group, of which *Scientific American* is a part, came out with *Principles of Biology*, **a** **new** **interactive, multimedia "book"** intended for university-level introductory biology classes that is accessible online using tablet computers, laptops, desktops and smartphones. *Principles of Biology* integrates text with videos, simulations, interactive exercises, illustrations and tests and also includes classic and current papers from *Nature* and related journals. Future titles in the life and physical sciences are in the works.

Marine ecologist David Johnston of Duke University and his colleagues have taken **a** **new** **Wikipedia-like approach**.

Their app, *Cachalot*, is available for free on the iPad and was created with the help of volunteers: marine scientists wrote it without charge from lecture notes, a computer science class designed it, and institutions, including the Woods Hole Oceanographic Institution, donated images and video. The project grew out of a class of Johnston's that focuses on large marine animals such as dolphins, turtles, seals and giant tube worms. Although writers are not paid for their contributions, their work does get peer-reviewed and published, thus making it potentially valuable when it **comes** **time** **for** **promotion** **or** **tenure**, he says.

Sharon Lynch, **a** **science** **education** **researcher** at George Washington University, says e-books such **as** **these** **may** **eventually** **become** **mainstream** **but** adds that research needs to be done on whether or not they are actually better than traditional textbooks. One such study is already under way at Nature Publishing Group: on some California State University campuses, students began biology on old textbooks, whereas other classes came in with *Principles of Biology*, so the company is doing side-by-side comparisons of how well students learned biology and how their attitudes toward science might differ, says Vikram Savkar, publishing director of Nature Education.

—Charles Q. Choi



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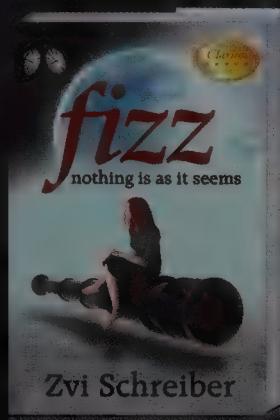
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SPACE

Swimming on Mars

The Red Planet may have once been home to an ocean

In the eyes of many
planetary scientists, the surface of Mars's northern hemisphere has long looked like it once contained an ocean. Now it is "sounding" that way, too.

A European spacecraft equipped with sounding radar that bounces radio waves off the Red Planet to investigate its makeup has identified what appear to be sedimentary deposits in the Martian north. The sediments, which could be mixed with ice, would represent the remains of a shallow ocean that existed some three billion years ago, according to a study published in January in *Geophysical Research Letters*.

The new research is based on a series of radar soundings by the MARSIS instrument on the European Space Agency's Mars Express orbiter, which has circled the Red Planet since 2003. "We mapped the intensity of the surface echo all over the planet," says lead study author Jérémie Mouginot, a geophysicist at the University of California, Irvine. In the Vastitas Borealis formation, a geologic deposit near the Martian north pole that has long been suspected of being sedimentary in origin, the radar reflectivity was quite low—lower than would be expected if the formation were volcanic rather than sedimentary.

Mouginot's interpretation is in sync with data obtained by another sounding radar on NASA's Mars Reconnaissance Orbiter, which surveyed the region a few years ago. That spacecraft's SHARAD instrument suggested that the Vastitas Borealis formation comprised a substantial sedimentary layer overlying volcanic plains.

Based on the extent of the sediments identified by Mars Express, the ocean would have overlain a large region of the



northern plains, though not for very long. Around three billion years ago Mars appears to have had enough geothermal activity to melt a large amount of groundwater and feed a shallow ocean, perhaps 100 meters deep. (There may also have existed an earlier ocean, Mouginot adds.) "I think what we had here is some episode of flash flooding or something like that that covered the northern plain," Mouginot says. But the environment would have been too cold and too dry to sustain a large body of water over geologic timescales. Within a million years or so the ocean would have refrozen and been buried underground or escaped as vapor.

The new radar data offer support—but not incontrovertible ground truth—for the long-held vision of an expansive body of water spread over the Martian north. "The ancient ocean hypothesis will take a while to prove to a high scientific standard because it's a bit buried, so to speak, today," says planetary scientist Norbert Schorghofer of the Institute for Astronomy at the University of Hawaii at Manoa, who was not part of the research team. And one can always wonder about additional interpretations of the radar echoes, which provide a relatively non-specific diagnosis of a given material. All the same, "it's another piece of evidence for an ancient ocean," he says. "I'm starting to believe it."

—John Matson

SCIENCE IN PICTURES

Seeing green? Scientists have long wondered how jumping spiders, such as this one, get visual information quickly and accurately enough to catch flies. In a study published in the journal *Science in Industry*, Takehito Nagata of Japan's Osaka City University and his colleagues reported that jumping spiders compare focused and unfocused images to perceive depth—with a color twist. The investigators knew that the two innermost layers of a jumping spider's own principal eyes (seen here as the two large eyes) are aimed toward green light. But they knew that light differently: the deepest layer focuses green light clearly, and the second layer receives defocused images. To test whether differences in the two layers were important for depth perception, Nagata's team shone green light on the spiders and rendered them with tiny illusions. The spiders made spot-on judgments. Yet when the team bathed the prey in red light that did not come in both wavelengths, the spiders consistently missed their prey.

—Katherine Lessor



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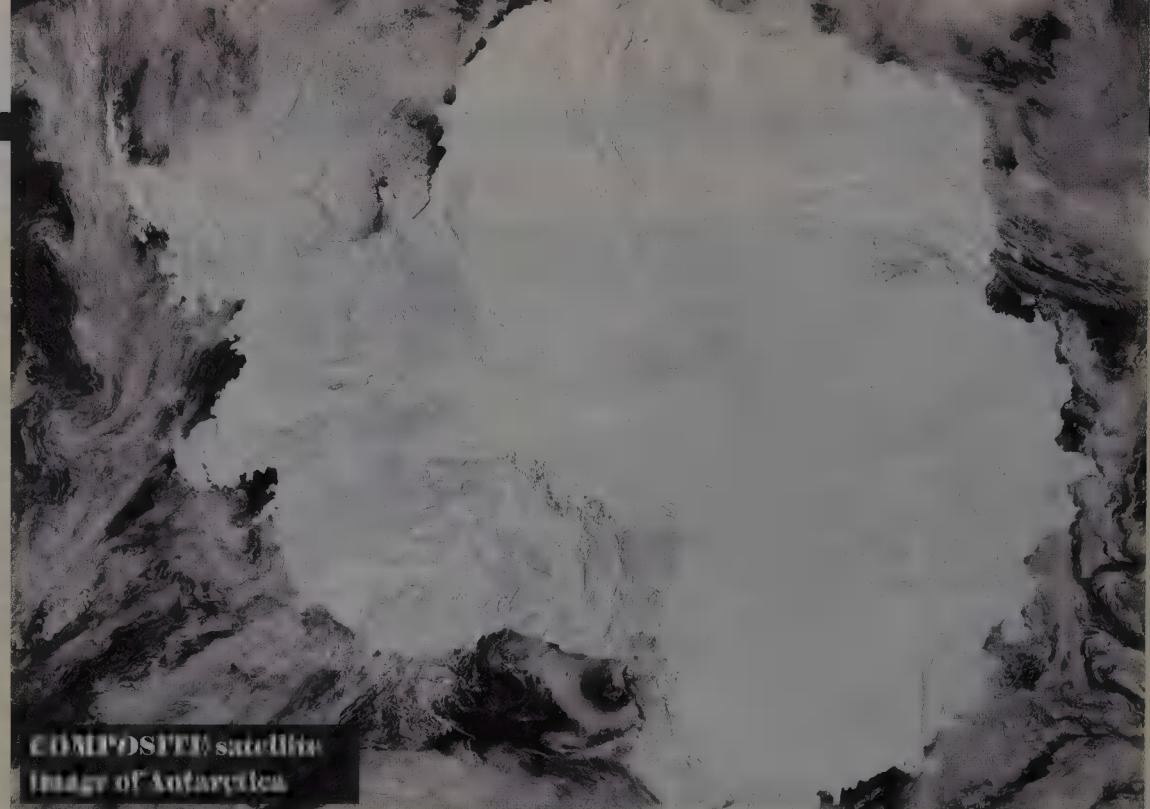
ECOLOGY

Cold Call

What will scientists find in Antarctica's ancient Lake Vostok?

Far below the surface of the central East Antarctic ice sheet is a body of water 160 miles long by 30 miles across known as Lake Vostok. The Vostok research station above it, for which it was named, was built by the former Soviet Union in 1957 and is now operated by Russia. Even by Antarctic standards, it is a brutal place, with the dubious honor of holding the record for the lowest measured temperature anywhere on the planet, a mind- (if not body-) numbing -129 degrees Fahrenheit (-89 degrees Celsius).

For the past 23 years, with a pause between 1998 and 2004, a hole has been gradually drilled down from this location into the ancient layers of ice. Hints that there could be a vast subsurface body of water arose in the 1950s and 1960s. Ground-penetrating radar later confirmed these suggestions, and Lake Vostok,



with its 1,300 cubic miles of liquid water, was revealed some two and a half miles below the ice.

It quickly became clear that this was an environment sealed away from the earth's surface, and although the water in the lake may itself be slowly changed out by the deep-ice dynamics of Antarctica, this process could take well over 10,000 years. It is also possible that hydrostatic sealing has kept the lake truly isolated for millions of years.

Devoid of light but likely bursting with supersaturated oxygen and other gases, Vostok has long been speculated to be a potential habitat for unique

ecosystems of extremophilic microbial life (and who knows what else). Despite the clear risks of contaminating what may be a pristine and fragile environment, Russian scientists have now, eight years after resuming, drilled to the top of the lake. Pressurized water from the lake rose through the borehole and froze, forming a plug. When scientists return this fall, they will remove the plug and check it for signs of life.

It is tremendously exciting, just as it is also tremendously worrying, that we will have messed up yet another irreplaceable ecosystem. If we're lucky,

however, what we will learn about the lost world of Lake Vostok may provide scientific impetus to get ourselves to one of the extraordinary subsurface oceans that exist elsewhere in our solar system, from the Jovian moons Europa and Ganymede to the geyser-spouting mysteries of distant Enceladus. It is possible that what is happening at the Vostok station today is the beginning of our next chapter in the search for life in the universe.

—Caleb A. Scharf

Adapted from Scharf's Life, Unbounded blog at blogs.ScientificAmerican.com/life-unbounded

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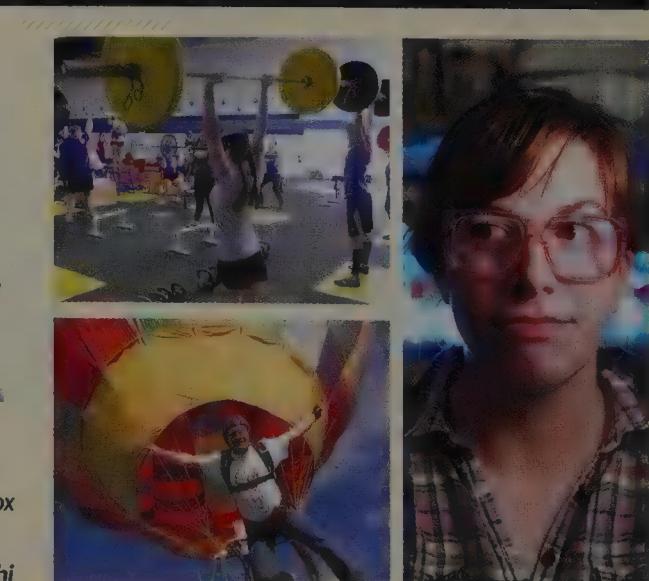
This Is What a Scientist Looks Like

A new Web site attempts to dispel a pervasive stereotype

If you ask middle school students what a scientist looks like, they will tell you he is an old white guy with crazy hair, glasses and a lab coat. More often than not, he is depicted inside and playing with chemicals. This stereotype is pervasive—and completely, totally wrong.

This is why I love the new Tumblr site [This Is What a Scientist Looks Like](http://lookslikescience.tumblr.com) at <http://lookslikescience.tumblr.com>. Scientists from all kinds of fields are asked to submit photographs of themselves and write a brief bit about who they are. The pictures are incredible; scientists are depicted everywhere from Antarctica to the tropics, on the tops of mountains or under the sea. The pics express personality, intelligence and even a little humor.

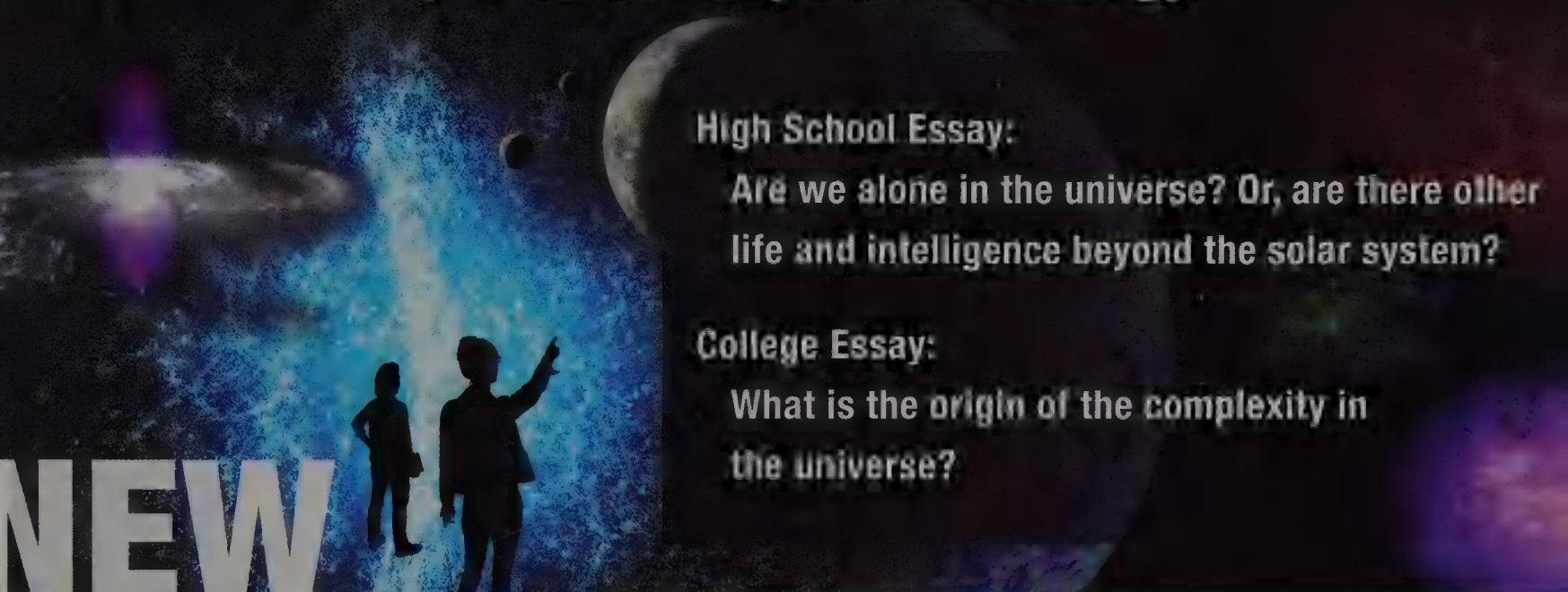
If you are a scientist, I strongly encourage you to add yourself. And if you are not, go check out what scientists really look like.



Adapted from Wilcox's Science Sushi blog at blogs.ScientificAmerican.com/science-sushi



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Maryn McKenna is a journalist, blogger and author of two books about public health. She writes about infectious diseases, global health and food policy.



Food Poisoning's Hidden Legacy

Most people think of foodborne illness as an unpleasant few days of fever and diarrhea, but for some there may be lifelong consequences

Colette Dziadul struggled for years to understand her daughter's joint problems. Dana, who is now 14 years old, complained from toddlerhood that her knees and ankles hurt. The aches kept her up at night, made her wake her parents to ask for painkillers and forced her to sit out school sports. Nevertheless, two pediatricians and an orthopedist diagnosed the problem as "growing pains" that would fade as she grew older.

Then, when Dana was 11, Dziadul participated in a survey about foodborne illness. The questionnaire came from an organization called Safe Tables Our Priority (now STOP Foodborne Illness), which was canvassing survivors of outbreaks for details of their recoveries. When she was three years old, Dana had spent two weeks in the hospital—one of 50 people sickened after eating cantaloupe that had been contaminated with *Salmonella*. Among the complications of infection that the survey listed were symptoms of a form of joint damage known as reactive arthritis.

Dziadul was dumbfounded. She found Dana a rheumatologist, who confirmed that the pain was caused by arthritis for which there was no other explanation. Then she went back into Dana's medical records. On Dana's 10th day in the hospital a nurse had recorded that the youngster was limping and complaining of joint pain. Could those long-forgotten symptoms have been the first sign of arthritis, starting as her body reacted to the *Salmonella* infection? "That there could be a connection between *Salmonella* and arthritis never crossed my mind," Dziadul says. "And it never crossed most of the doctors' minds."

It is a scary idea that food poisoning—which we think of as lasting just a few days—could instead have lifelong aftereffects. The incidence of such "sequelae," in medical parlance, has been thought to be low, but not many researchers studied the problem until recently. New findings by several scientific teams suggest the phenomenon is more common than anyone thought.

A COMMON PROBLEM?

Foodborne disease has an enormous public health impact even if you count only the initial, acute episodes of illness. The Centers for Disease Control and Prevention estimated in 2011 that



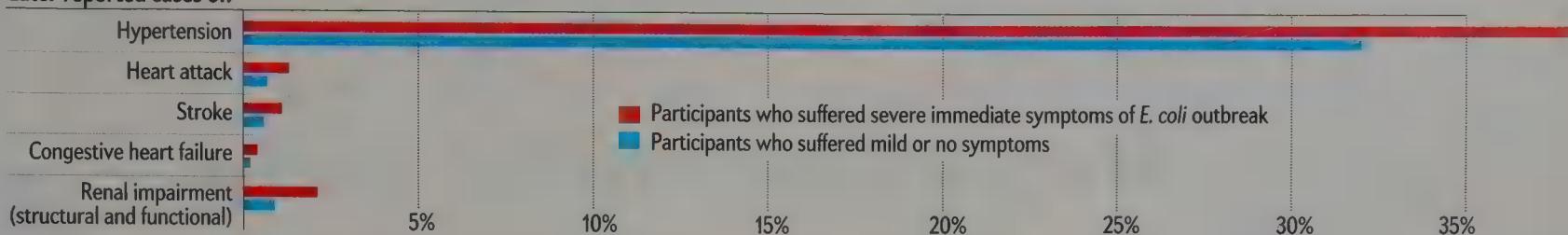
the U.S. sees 48 million illnesses, 128,000 hospitalizations and 3,000 deaths every year from foodborne organisms. (The European Union had 48,964 cases and 46 deaths in 2009, the most recent year tallied.) The U.S. Department of Agriculture's Economic Research Service calculates the cost of foodborne illnesses just from bacterial infection to be at least \$6.7 billion, counting medical care, premature deaths and lost productivity. Researchers who attempt to track chronic effects say that the actual bill is much higher.

"People don't understand the full consequences of foodborne disease," says Kirk Smith of the Minnesota Department of Health, which lends its investigators around the U.S. "They think you get diarrhea for a few days and then you are better. They don't understand that there is a whole range of chronic sequelae. And although any of them may not be common individually, when you put them together they add up to a lot."

Long-term consequences are not limited to individuals who were hospitalized, as Dana was. They have also been recorded in people who experienced what seemed to be minor bouts of fever, vomiting or diarrhea. The consequences include reactive arthritis, urinary tract problems and damage to the eyes after *Salmonella* and *Shigella* infections; Guillain-Barré syndrome and ulcerative colitis (a chronic bowel inflammation) after *Campylobacter* infection; and kidney failure and diabetes after infection with *Escherichia coli* O157:H7. Those organisms are very com-

Long-Term Health Effects of Bacterial Infection

Later reported cases of:



Aftermath: For six years investigators followed 4,561 residents of a small Canadian town whose water supply had been contaminated with *E. coli* O157:H7. The researchers found statistically significant increases in heart and kidney problems among study participants who had suffered from diarrhea or vomiting during the outbreak compared with those who exhibited mild or no symptoms.

mon: federal investigators have identified them in meat, milk, poultry, eggs, seafood, fruit, vegetables and even processed foods.

As researchers look back at foodborne outbreaks, they are not only confirming that these complications appear in survivors but adding to the list of illnesses that may occur. A survey of 101,855 residents of Sweden who were made sick by food between 1997 and 2004 found, for instance, that they had higher-than-normal rates of aortic aneurysms, ulcerative colitis and reactive arthritis. A review of a major provincial health database in Australia revealed that people there who contracted any bacterial gastrointestinal infection were 57 percent more likely to develop either ulcerative colitis or Crohn's disease, another chronic bowel condition, than people born in the same place and era who had not had such infections. And several years after a 2005 outbreak of *Salmonella* in Spain, 65 percent of 248 victims said they had developed joint or muscle pain or stiffness, compared with 24 percent of a control group who were not affected by the outbreak.

Few comprehensive analyses have been conducted in the U.S. Traditionally, food-related investigations have aimed at finding and interviewing victims during the outbreak, Smith says. Because acute illness lasts a couple of weeks at most, little attention has been paid to keeping track of victims afterward—something that might be very complicated because they may go to different doctors and even live in different states.

One of the U.S. studies, published in 2008, traced victims of foodborne illness in Minnesota and Oregon between 2002 and 2004. Researchers determined whom to contact based on records collected by a CDC surveillance project known as the Foodborne Diseases Active Surveillance Network (FoodNet), which collects reports of lab-confirmed infections caused by 10 different organisms. Out of 4,468 victims, 575 (13 percent) reported later symptoms that matched reactive arthritis, although most—unlike Dana—were never diagnosed by a specialist.

The link between foodborne illness and long-term health consequences could be a coincidence, although advocates say that the chances are remote. A better way to prove the connection would be to identify victims when they first become ill and track them for years thereafter, a research arrangement called a prospective study. There are a few such studies worldwide, and a recently concluded one—the only one to take place in North America—was stunning and persuasive.

In May 2000 the drinking water in Walkerton, Ont., became contaminated with *E. coli* O157 after heavy rains washed manure

from farm fields into its aquifer. More than 2,300 people, about half the town's population, developed fever and diarrhea soon afterward. In 2002 the Ontario government funded the Walkerton Health Study to assess any health effects that might persist among the victims. In 2010 the study published its findings: compared with residents who did not get very sick, those who endured several days of diarrhea during the outbreak had a 33 percent greater likelihood of developing high blood pressure, a 210 percent greater risk of heart attack or stroke, and a 340 percent greater risk of kidney problems in the eight years following the outbreak.

Those outcomes were not limited to people who developed the most serious consequences of *E. coli* O157 infection. Even Walkerton residents with milder symptoms experienced circulatory problems that would not have been linked to *E. coli* without the prospective monitoring. That discovery suggests how common the late-onset effects of *E. coli* infection might be, says William F. Clark, the study's leader and a professor of nephrology at the University of Western Ontario. Clark recommends that survivors of such illnesses have their blood pressure checked every year and their kidney function checked every two or three years.

Given how few scientists have studied the issue, most of the problems have come to light thanks to patient advocacy groups. STOP's original survey, in which Colette Dziadul participated, collected first-person accounts from patients. It was followed by a 2009 white paper from the nonprofit Center for Foodborne Illness Research and Prevention, which unearthed research on long-term sequelae that were buried in the medical literature.

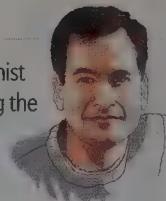
That group now has a grant from the U.S. Food and Drug Administration to research how best to study the frequency of persistent aftereffects. Advocates want public health agencies to create better mechanisms for identifying and tracking victims, and like Clark, they think victims should be connected as soon as possible to preventive medical care.

"We want to establish the true burden of disease because that is what policy makers use to decide what is a public health priority," says Barbara Kowalcyk, the center's co-founder. "As long as we focus only on the acute form of foodborne illness and not the long-term health consequences, we'll underestimate how significant a problem this is." ■

SCIENTIFIC AMERICAN ONLINE

Comment on this article at ScientificAmerican.com/apr2012

David Pogue is the personal-technology columnist for the *New York Times*. He is the host of "Hunting the Elements" on *NOVA*, which airs April 4 on PBS.



Technology's Friction Problem

Make buying, voting and losing weight easier by blasting away unnecessary steps



A few months back I was at the main Apple Store in New York City. I wanted to buy a case for my son's iPod touch—but it was December 23. The crowds were so thick, I envied sardines.

Fortunately, I knew something that most of these people didn't: I could grab an item off the shelf, scan it with my iPhone and walk right out. Thanks to the free Apple Store app, I didn't have to wait in line or even find an employee. The purchase was instantly billed to my Apple account. I was in and out of there in two minutes.

Apple, in other words, has reached new heights in reducing friction—which benefits it as much as its customers.

Friction is a hassle. Steps. Process. And in this increasingly technified world, there is still a surprising amount of red tape—and few examples of push back. We stress about things like price, storage and processor speed, instead of beauty, elegance and low friction.

Why do some stores still make us sign credit-card slips? There is no legal or bank requirement to collect signatures. That bit of friction was originally intended as a security measure—but when is the last time you saw a clerk compare your signature with one on the back of the card?

Why, in this day and age, are we still typing in our address and credit-card details into Web forms, over and over again? Companies like Apple and Amazon have figured it out. Low friction means more sales. Apple has its app; Amazon has its 1-Click Buy

button. You don't have to enter any extra information. You see something you want, you click, and you've just bought it.

Every Web site that makes you fill in a form, or wait for a confirmation e-mail, or take some test to prove that you are human is adding friction—and losing sales. All of us, sooner or later, will wind up sitting there with a comment to make or a product to buy, see how many hoops we have to jump through and then back out: "Oh, forget it—not worth it."

Actually, low friction doesn't just mean more sales. It means more of *any* behavior you're trying to encourage. Take, for example, the right to vote.

The formula for predicting someone's likelihood to vote is something like $PB + D > C$, where P is the probability that your vote will make a difference, B is the benefit to you if your candidate wins, D is the gratification you get from voting, and C is friction—the hassle of registering to vote, then getting to the polling place, standing in line, and so on. Clearly, lowering the friction would increase turnout.

Imagine if we could register and vote online—or vote by making a few taps in a phone app. Voter turnout would likely skyrocket. And that would make for a real democracy. (Fear of manipulation is supposedly the reason we're not there yet. But we could get there if we really wanted to.)

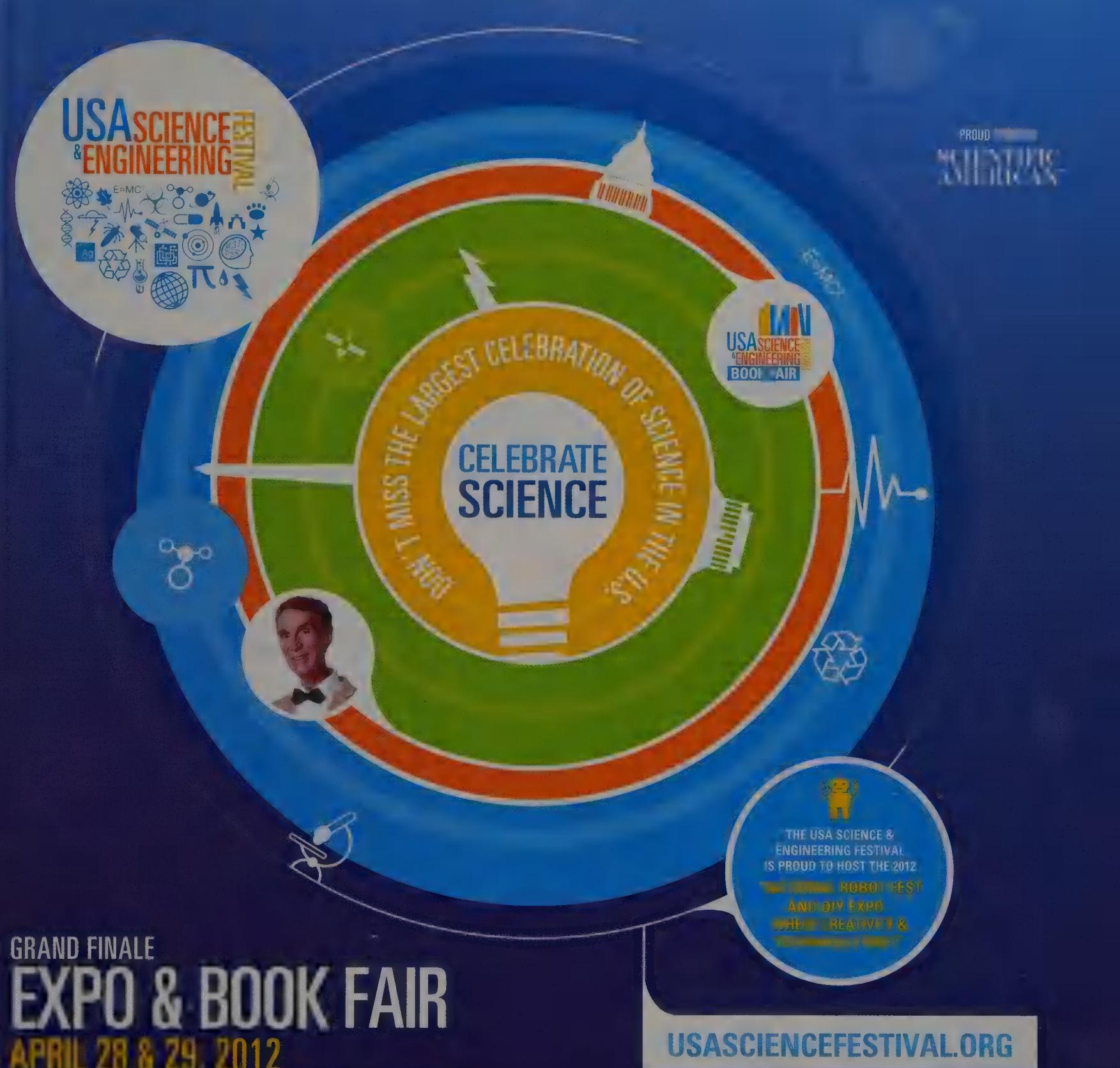
Or what about the obesity epidemic? We've tried almost every solution under the sun—except reducing friction. You can buy coffee with a tap on a Starbucks app, so why not healthy foods? Why can't you get an apple, banana or bag of baby carrots in more vending machines or from a market with an app tap? Eating right still takes more effort than eating junk. Change the friction coefficient, and you change the game.

Next time you're shopping for a digital camera, don't ask how many megapixels it has. Ask how many steps it takes to turn on the manual focus. When you buy a laptop, don't just care about its screen size; care about how many touch tones are required to get you to tech support. When you buy a phone, see how many taps it takes to e-mail a photo.

And if you're on the other side of the table—if you're the vendor—don't just figure out how to attract customers. Figure out how to eliminate the friction you present to them. ■

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Read about four wins over friction at ScientificAmerican.com/apr2012/pogue



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HUMAN EVOLUTION

First of Our Kind

Sensational fossils from South Africa spark debate over how we came to be human

By Kate Wong

IN BRIEF

The origin of our genus, *Homo*, is one of the biggest mysteries facing scholars of human evolution.

Based on the meager evidence available, scientists have surmised that *Homo* arose in East

Africa, with Lucy's species, *Australopithecus afarensis*, giving rise to the founding member of our lineage, *Homo habilis*.

Recently discovered fossils from a site northwest of Johannesburg, South Africa, could up-

end that scenario. The fossils represent a previously unknown species of human with an amalgam of australopithecine and *Homo* traits that suggest to its discoverers that it could be the ancestor of *Homo*.

NEW HUMAN SPECIES from South Africa—*Australopithecus sediba*—has been held up as the ancestor of our genus, *Homo*.

S

OMETIME BETWEEN THREE MILLION AND TWO million years ago, perhaps on a primeval savanna in Africa, our ancestors became recognizably human. For more than a million years their australopithecine predecessors—Lucy and her kind, who walked upright like us yet still possessed the stubby legs, tree-climbing hands and small brains of their ape forebears—had thrived in and around the continent's forests and woodlands. But their world was changing. Shifting climate favored the spread of open grasslands, and the early australopithecines gave rise to new lineages. One of these offshoots evolved long legs, toolmaking hands and an enormous brain. This was our genus, *Homo*, the primate that would rule the planet.

by legs, tree-climbing hands and small brains of their ape forebears—had thrived in and around the continent's forests and woodlands. But their world was changing. Shifting climate favored the spread of open grasslands, and the early australopithecines gave rise to new lineages. One of these offshoots evolved long legs, toolmaking hands and an enormous brain. This was our genus, *Homo*, the primate that would rule the planet.

For decades paleoanthropologists have combed remote corners of Africa on hand and knee for fossils of *Homo*'s earliest representatives, seeking to understand the details of how our genus rose to prominence. Their efforts have brought only modest gains—a jawbone here, a handful of teeth there. Most of the recovered fossils instead belong to either ancestral australopithecines or later members of *Homo*—creatures too advanced to illuminate the order in which our distinctive traits arose or the selective pressures that fostered their emergence. Specimens older than two million years with multiple skeletal elements preserved that could reveal how the *Homo* body plan came together eluded discovery. Scientists' best guess is that the transition occurred in East Africa, where the oldest fossils attributed to *Homo* have turned up, and that *Homo*'s hallmark characteristics allowed it to incorporate more meat into its diet—a rich source of calories in an environment where fruits and nuts had become scarce. But with so little evidence to go on, the origin of our genus has remained as mysterious as ever.

Lee Berger thinks he has found a big piece of the puzzle. A paleoanthropologist at the University of the Witwatersrand in Johannesburg, South Africa, he recently discovered a trove of fossils that he and his team believe could revolutionize researchers' understanding of *Homo*'s roots. In the white-walled confines of room 210 at the university's Institute for Human Evolution, he watches as Bernard Wood of George Washington University paces in front of the four plastic cases that have been removed from their fireproof safe and placed on a table clothed in royal blue velvet. The foam-lined cases are open, revealing the nearly two-million-year-old fossils inside. One holds pelvis and leg bones. Another contains ribs and vertebrae. A third displays arm bones and a clavicle. And a fourth houses a skull. On a counter opposite the table, more cases hold a second partial skeleton,

including a nearly complete hand.

Wood, a highly influential figure in the field, pauses in front of the skull and leans in for a closer look. He strokes his beard as he considers the dainty teeth, the grapefruit-size braincase. Straightening back up, he shakes his head. "I'm not often at a loss for

words," he says slowly, almost as if to himself, "but wow. Just wow."

Berger grins. He has seen this reaction before. Since he unveiled the finds in 2010, scientists from all over the world have been flocking to his lab to gawk at the breathtaking fossils. Based on the unique anatomical package the skeletons present, Berger and his team assigned the remains to a new species, *Australopithecus sediba*. They furthermore propose that the combination of primitive *Australopithecus* traits and advanced *Homo* traits evident in the bones qualifies the species for a privileged place on the family tree: as the ancestor of *Homo*. The stakes are high. If Berger is right, paleoanthropologists will have to completely rethink where, when and how *Homo* got its start—and what it means to be human in the first place.

THE ROAD NOT TAKEN

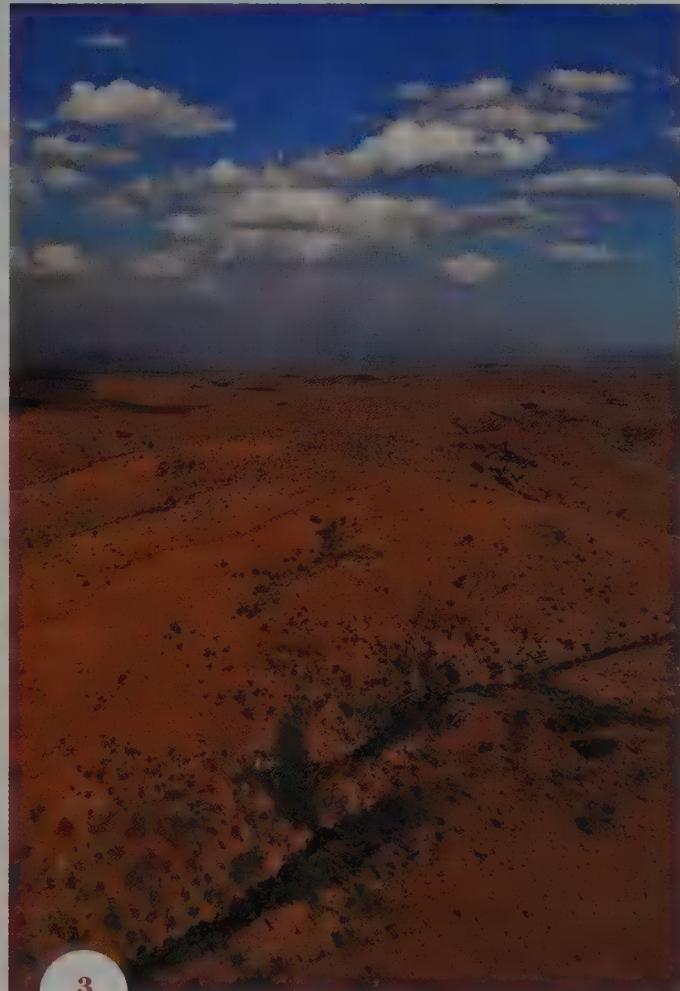
IN THE MIDDLE of the rock-strewn dirt road that winds through the John Nash Nature Reserve, Berger brings the Jeep to a halt and points to a smaller road that branches right. For 17 years he had made the 40-kilometer trip northwest from Johannesburg to the 9,000-hectare parcel of privately owned wilderness and driven past this turnoff, continuing along the main road, past the resident giraffes and warthogs and wildebeests, to a cave he was excavating just a few kilometers away called Gladysvale. In 1948 American paleontologists Frank Peabody and Charles Camp came to this area to look for fossils of hominins (modern humans and their extinct relatives) on the advice of famed South African paleontologist Robert Broom, who had found such fossils in the caves of Sterkfontein and Swartkrans, eight kilometers away. Peabody suspected that Broom had intentionally sent them on a wild goose chase, so unimpressed was he with the sites here. Little did Berger or the expeditioners before him know that had they only followed this smaller path—one of



1



2



3

LEE BERGER (left) and Meshack Kgasi (right) inspect the miners' pit at the Malapa site, where Berger discovered *Australopithecus sediba* (1). Blocks of concretelike calcified clastic sediment dislodged by miners will be CT-scanned to see if they contain fossils (2). View captures the valleys in and around the Malapa area, northwest of Johannesburg in South Africa (3).

several miners' tracks used in the early 1900s to cart the limestone that built Johannesburg from quarries out to the main road—they would have made the discovery of a lifetime.

Berger, now 46 years old, never imagined he would find something like *A. sediba*. Although he thought *Homo* might have had roots in South Africa instead of East Africa, he knew the odds of making a big find were slim. Hominin fossils are extremely rare, so "you don't have any expectations," he reflects. What is more, he was focused on the so-called Cradle of Humankind, an already intensively explored region whose caves had long been yielding australopithecines generally considered to be more distantly related to *Homo* than the East African australopithecines seemed to be. And so Berger continued to toil at Gladysvale day after day, year after year. Because he found little in the way of hominins among the millions of animal fossils there—just scraps of a species called *A. africanus*—he busied himself with another goal: dating the site. A critical problem with interpreting the South African hominin fossils was that scientists had not yet figured out how to reliably determine how old they were. In East Africa, hominin fossils come from sediments sandwiched between layers of volcanic ash that blanketed the landscape during long-ago eruptions. Geologists can ascertain how old an ash layer is by analyzing its chemical "fingerprint." A fossil that originates from a layer of sediment that sits in between two volcanic ashes is thus intermediate in age between those two ashes. The cave sites in the Cradle of Humankind lack volcanic ashes. Through his 17 years of trial and error at Gladysvale, however, Berger and his colleagues hit on techniques that circumvented the problem of not having ash to work with.

Those techniques would soon come in very handy. On August 1, 2008, while surveying the reserve for potential new fossil sites in the area that he had identified using Google Earth, Berger turned right on the miners' track he had passed by for 17 years and followed it to a three- by four-meter hole in the ground blasted by the miners. Eyeballing the site, he found a handful of animal fossils—enough to warrant a trip back for a closer look. He returned on August 15 with his then nine-year-old son, Matthew, and dog, Tau. Matthew took off into the bush after Tau, and within minutes he shouted to his father that he had found a fossil. Berger doubted it was anything important—probably just an antelope bone—but in a show of fatherly support, he made his way over to inspect the find. There, protruding from a dark hunk of rock nestled in the tall grass by the corpse of a lightning-struck tree, was the tip of a collarbone.

As soon as Berger laid eyes on it, he knew it belonged to a hominin. In the months that followed he found more of the clavicle's owner, along with another partial skeleton, 20 meters away in the miners' pit. To date, Berger and his team have recovered more than 220 bones of *A. sediba* from the site—more than all the known early *Homo* bones combined. He christened the site *Malapa*, meaning "homestead" in the local Sesotho language. Using the approaches honed at Gladysvale, the geologists on Berger's team would later date the remains with remarkable precision to 1.977 million years ago, give or take 2,000 years.

A PATCHWORK PREDECESSOR

THAT THE MALAPA FOSSILS include so many body parts is important because it means they can offer unique insights into the order in which key *Homo* traits appeared. And what they show very

clearly is that quintessentially human features did not necessarily evolve as a package deal, as was thought. Take the pelvis and the brain, for example. Conventional wisdom holds that the broad, flat pelvis of australopithecines evolved into the bowl-shaped pelvis seen in the bigger-brained *Homo* to allow delivery of babies with larger heads. Yet *A. sediba* has a *Homo*-like pelvis with a broad birth canal in conjunction with a teeny brain—just 420 cubic centimeters, a third of the size of our own brain. This combination shows brain expansion was not driving the metamorphosis of the pelvis in *A. sediba*'s lineage.

Not only do the *A. sediba* fossils mingle old and new versions of general features, such as brain size and pelvis shape, but the pattern repeats at deeper levels, like an evolutionary fractal. Analysis of the interior of the young male's braincase shows that the brain, while small, possessed an expanded frontal region, indicating an advanced reorganization of gray matter; the adult female's upper limb pairs a long arm—a primitive holdover from a tree-dwelling ancestor—with short, straight fingers adapted to making and using tools (although the muscle markings on the bones attest to powerful, apelike grasping capabilities). In some instances, the juxtaposition of old and new is so improbable that had the bones not been found joined together, researchers would have interpreted them as belonging to entirely different creatures. The foot, for instance, combines a heel bone like an ancient ape's with an anklebone like *Homo*'s, according to Malapa team member Bernard Zipfel of the University of the Witwatersrand. It is as if evolution was playing Mr. Potato Head, as Berger puts it.

The extreme mosaicism evident in *A. sediba*, Berger says, should be a lesson to paleoanthropologists. Had he found any number of its bones in isolation, he would have classified them differently. Based on the pelvis, he could have called it *H. erectus*. The arm alone suggests an ape. The anklebone is a match for a modern human's. And like the blind men studying the individual parts of the elephant, he would have been wrong. "*Sediba* shows that one can no longer assign isolated bones to a genus," Berger asserts. That means, in his view, finds such as a 2.3-million-year-old upper jaw from Hadar, Ethiopia, that has been held up as the earliest trace of *Homo* cannot safely be assumed to have belonged to the *Homo* line.

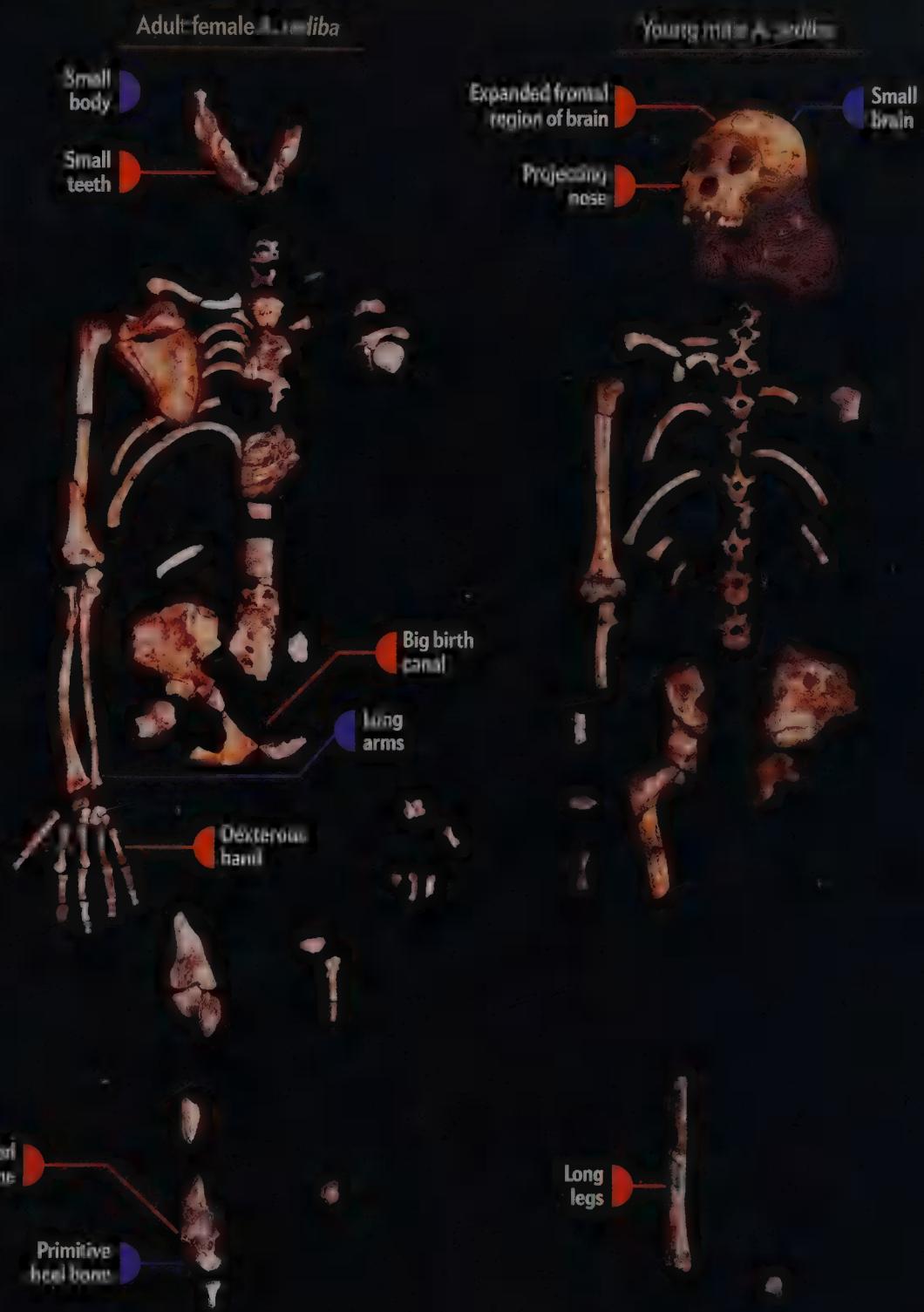
Taking that jaw out of the running would make *A. sediba* older than any of the well-dated *Homo* fossils but still younger than *A. afarensis*, putting it in pole position for the immediate ancestor of the genus, Berger's team contends. Furthermore, considering *A. sediba*'s advanced features, the researchers propose that it could be specifically ancestral to *H. erectus* (a portion of which is considered by some to be a different species called *H. ergaster*). Thus, instead of the traditional view in which *A. afarensis* begat *H. habilis*, which begat *H. erectus*, he submits that *A. africanus* is the likely ancestor of *A. sediba*, which then spawned *H. erectus*.

If so, that arrangement would relegate *H. habilis* to a dead-end side branch of the human family tree. It might even kick *A. afarensis*—long considered the ancestor of all later hominins, including *A. africanus* and *Homo*—to the evolutionary curb, too. Berger points out that *A. sediba*'s heel is more primitive than that of *A. afarensis*, indicating that *A. sediba* either underwent an evolutionary reversal toward a more primitive heel or that it descended from a different lineage than the one that includes *A. afarensis* and *A. africanus*—one that has yet to be discovered.

"In the South, we have a saying: 'You dance with the girl you

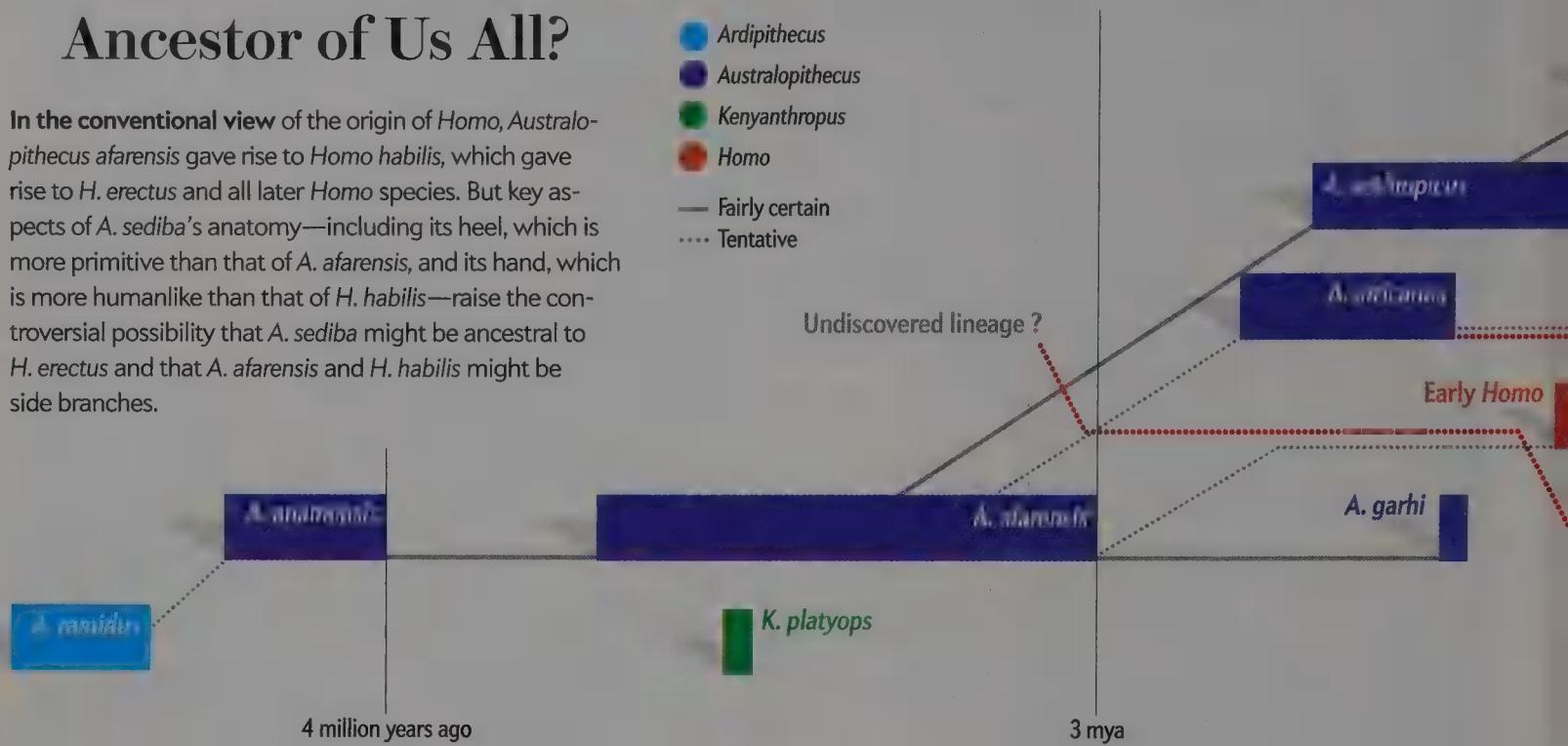
Mix and Match

Australopithecus sediba skeletons exhibit a totally unexpected mix of australopithecine and *Homo* traits, representative examples of which are shown here. Previously, scientists thought that *Homo* features such as short arms and dexterous hands evolved in lockstep, but *A. sediba* shows that they emerged piecemeal—in this case marrying long, tree-climbing arms with hands whose short fingers and long thumb would have enabled a humanlike precision grip. *A. sediba*'s particular blend suggests to Berger's team that it descended from *A. africanus* or an unknown lineage and gave rise directly to *H. erectus*.

*H. erectus**A. africanus**sediba*

Ancestor of Us All?

In the conventional view of the origin of *Homo*, *Australopithecus afarensis* gave rise to *Homo habilis*, which gave rise to *H. erectus* and all later *Homo* species. But key aspects of *A. sediba*'s anatomy—including its heel, which is more primitive than that of *A. afarensis*, and its hand, which is more humanlike than that of *H. habilis*—raise the controversial possibility that *A. sediba* might be ancestral to *H. erectus* and that *A. afarensis* and *H. habilis* might be side branches.



brought,’’ quips Berger, who grew up on a farm in Sylvania, Ga. ‘‘And that is what paleoanthropologists have been doing’’ in trying to piece together the origin of *Homo* from the fossils that have turned up in East Africa. ‘‘Now we have to recognize there is more potential out there,’’ he states. Maybe the East Side story of human origins is wrong. The traditional view of South Africa’s oldest hominin fossils is that they represent a separate evolutionary experiment that ultimately fizzled out. *A. sediba* could turn the tables and reveal, in South Africa, another lineage, the one that ultimately gave rise to humankind as we know it (indeed, *sediba* is the Sesotho word for ‘‘fountain’’ or ‘‘wellspring’’).

William Kimbel of Arizona State University, who led the team that found the 2.3-million-year-old jawbone in Ethiopia, is having none of it. The idea that one needs a skeleton to classify a specimen is a “nonsensical argument,” he retorts. The key is to find pieces of anatomy that contain diagnostic traits, he says, and the Hadar jaw has features clearly linking it to *Homo*, such as the parabolic shape formed by its tooth rows. Kimbel, who has seen the Malapa fossils but not studied them in depth, finds their *Homo*-like traits intriguing, although he is not sure what to make of them. He scoffs at the suggestion that they are directly ancestral to *H. erectus*, however. “I don’t see how a taxon with a few characteristics that look like *Homo* in South Africa can be the ancestor [of *Homo*] when there’s something in East Africa that is clearly *Homo* 300,000 years earlier,” he declares, referring to the jaw.

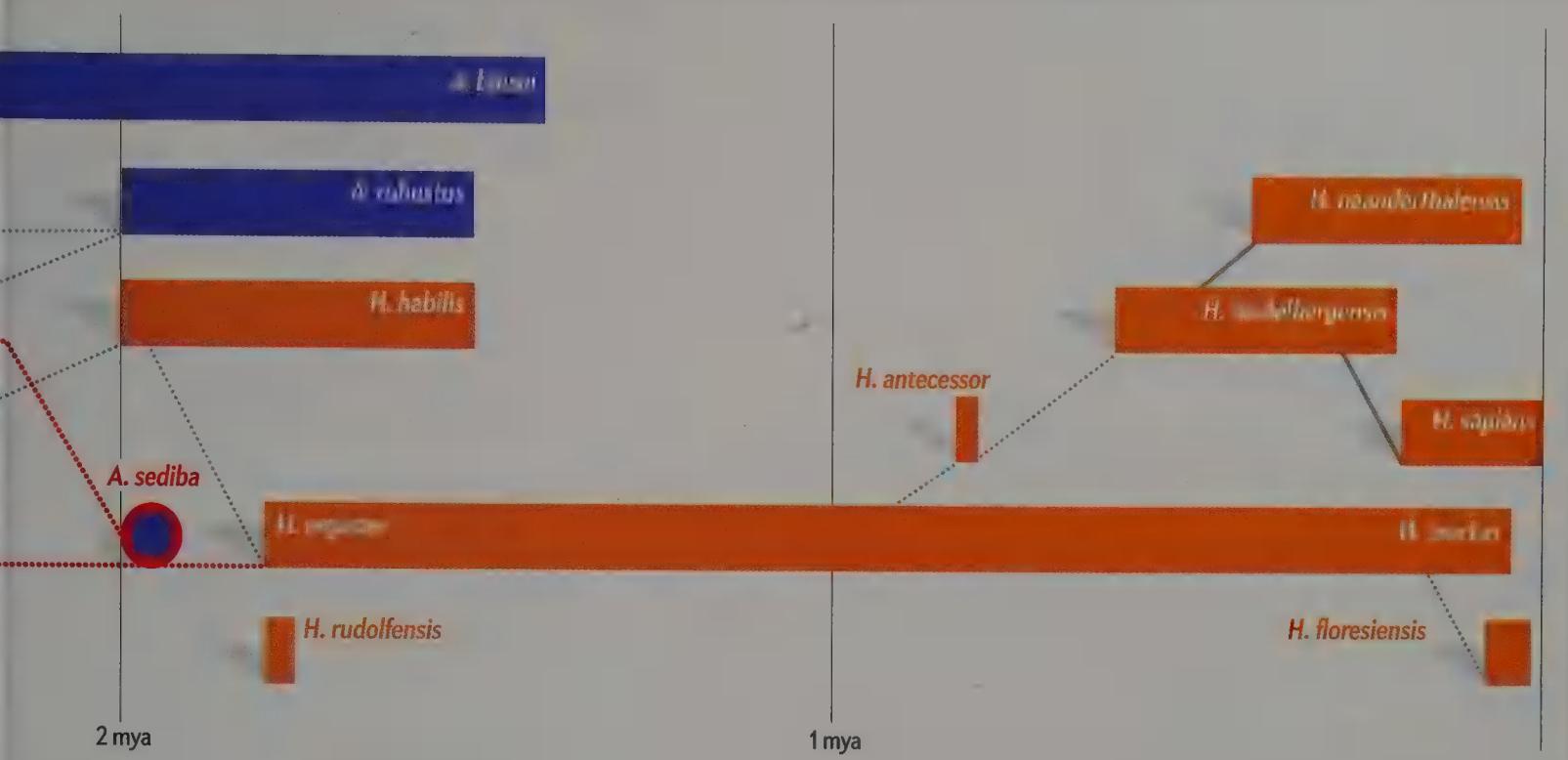
Kimbel is not alone in rejecting the argument for *A. sediba* as the rootstock of *Homo*. “There are too many things that do not fit, particularly the dates and geography,” comments Meave Leakey of the Turkana Basin Institute in Kenya, whose own research has focused on fossils from East Africa. “It is much more likely that the South African hominins are a separate radiation that took place in the south of the continent.”

René Bobe of George Washington University says that if the *A. sediba* remains were older—say, around 2.5 million years old—

they might make for a plausible *Homo* ancestor. But at 1.977 million years old, they are just too primitive in their overall form to be ancestral to fossils from Kenya's Lake Turkana region that are just a tad younger yet have many more indisputable *Homo* traits. Berger counters that *A. sediba* almost certainly existed as a species before the Malapa individuals. Bobe and others maintain that such information is not currently known. "Paleoanthropologists tend to think of the fossils they find as being in a key position within the [hominin] phylogenetic tree, and in many cases that's unlikely to be the situation," Bobe observes. From a statistical standpoint, "if you have [hominin] populations distributed across Africa, evolving in complex ways, why would the one you find be the ancestor?"

Berger has found a sympathetic ear in Wood, who says Berger is "absolutely right" that *A. sediba* demonstrates that isolated bones do not predict what the rest of the animal looks like. *A. sediba* shows that the combinations of traits evident from previous fossil discoveries do not exhaust the possibilities, Wood remarks. But he does not endorse the suggestion that *A. sediba* is the ancestor of *Homo*. "There are not many characters linking it to *Homo*," he notes, and *A. sediba* may have evolved those traits independently from the *Homo* lineage. "I just think *sediba* has got too much to do in order to evolve into [*erectus*].” Wood says.

Resolution of the issue of where *A. sediba* belongs in our family tree is hampered by the lack of a clear definition of the genus *Homo*. Coming up with one, however, is a taller order than it might seem. With so few specimens from the transition period, and most of them being scraps, identifying those features that first distinguished *Homo* from its australopithecine forebears—those traits that made us truly human—has proved challenging. The skeletons from Malapa expose just how vexing the situation is: they are so much more complete than any early *Homo* specimen that it is very difficult to compare them with anything. “*Sediba* may force us to come up with a definition,” Berger says.



ALL IN THE DETAILS

WHATEVER THE POSITION of the Malapa fossils in the family tree, they are poised to provide researchers with the most detailed portrait yet of an early hominin species, in part because they make up multiple individuals. In addition to the juvenile male and the adult female, the two most complete specimens, Berger's team has collected bones representing another four individuals, including a baby. Populations are incredibly rare in the human fossil record, and the individuals at Malapa have the added benefit of peerless preservation. Hominin bones that virtually never survive the ravages of deep time have turned up here: a paper-thin shoulder blade, the delicate sliver that is the first rib, pea-size finger bones, vertebrae with spiny projections intact. And a number of bones that were previously known only from fragments are complete. Before the discovery of Malapa, paleoanthropologists did not have a single complete arm from an early hominin, meaning that the limb lengths that are used to reconstruct such essential behaviors as locomotion are estimates. Even Lucy—the most complete hominin of such antiquity back when she was found in 1974—is missing significant chunks of her arm and leg bones. In the adult female from Malapa, in contrast, virtually the entire upper limb is preserved—from shoulder blade to hand. Only the very last digits of some of her fingers and some wristbones are missing, and Berger expects to find those—and the rest of the bones of both skeletons when he excavates the site (thus far the team has only collected bones visible from the surface, rather than systematically digging for buried material). From this evidence, researchers will be able to reconstruct how *A. sediba* matured, how it moved around the landscape and how members of the population varied from one another, among other things.

It is not only the bones that promise to tell new tales. Malapa has also yielded some other materials that could literally flesh out researchers' understanding of *A. sediba*. Paleontologists have

long thought that during the fossilization process, all of an organism's organic components—such as skin, hair, organs, and so forth—are lost to decomposition, leaving behind only mineralized bone. But when Berger saw a CT scan of the skull of the young male, he noticed a place on the crown where there appeared to be an air space between the surface of the fossil and the contour of the actual bone. Examining the spot more closely, he observed a distinctive pattern on the surface that looked like the structural components of skin. He is now conducting extensive tests to determine whether the odd-looking patch on the male's crown and another on the female's chin—and similar patches on antelope bones from the site—are in fact skin.

Preserved skin, if confirmed as such, could reveal *A. sediba*'s coloring and the density and patterning of its hair. Such evidence could also show the distribution of sweat glands—information that would provide insights into how well the species was able to regulate its body temperature, which in turn would have affected how active it was. Sweat glands could additionally offer clues to brain evolution: an effective means of keeping cool was a prerequisite for the emergence of large brain size—a trademark characteristic of *Homo*—because brains are temperature-sensitive. And if organic material is present, Berger might even be able to obtain DNA from the remains. Currently the oldest hominin DNA to have been sequenced is 100,000-year-old DNA from a Neandertal. But because the preservation conditions at Malapa were apparently exceptional, Berger has some hope of getting genetic information from the much older *A. sediba* specimens. In that event, scientists might be able to determine whether the adult female and young male really were mother and son, as has been suggested, and how, if at all, the other hominins at the site fit in. Moreover, such a discovery would prompt researchers at other early hominin sites to test for DNA, which, if successful, could settle debates over how the various hominin species were related.



SYNCHROTRON X-RAY SCANNING of the skull of the young male *A. sediba* enabled detailed reconstruction of the brain (pink), which exhibits advanced reorganization in the frontal lobes despite being little larger than a chimpanzee's brain.

Preservation of organic remains would be a first in hominin paleontology, and the Malapa team knows it will need extraordinary evidence to persuade the research community of such a claim. Thus far, however, the test results support the hypothesis, and Berger thinks the odds are very good that future analyses will bear it out. After all, similar claims have been made for organic material from dinosaur bones, and those are tens of millions of years older than the Malapa fossils. Organic preservation in hominin assemblages might even be fairly common, he suggests—it is just that no one ever thought to look for it.

Another thing no one thought to look for in a hominin this old? Tartar. The surfaces of the young male's molar teeth bear dark brown stains. Fossil preparators typically clean off the

teeth when readying hominin remains for study. But it occurred to Berger that the stains might actually be the same gunk we modern humans fend off with toothbrushes and pilgrimages to the dentist. Ancient tartar would provide valuable insights into the evolution of the hominin diet.

Previous studies of what early humans ate have looked at carbon isotope ratios in teeth, which can indicate whether an animal dined on so-called C3 plants, such as trees and shrubs, or C4 plants, such as certain grasses and sedges—or, in the case of carnivorous species, preyed on animals that ate those plant foods—or some combination thereof over its lifetime. Such evidence is indirect and nonspecific. Tartar, in contrast, is the remnants of the food itself. The team is currently studying tiny

silica crystals called phytoliths that are embedded in the tartar. Phytoliths come from plants, and some plants make species-specific forms of the crystals. Studies of these phytoliths can thus reveal exactly which kinds of plants an animal ate just before it died. By analyzing the isotope ratios, phytoliths and wear marks on *A. sediba*'s teeth that can signal whether an animal was chewing harder or softer foods in the weeks before it perished, the team should be able to glean a wealth of subsistence data. And because the researchers have bones from *A. sediba* individuals across a range of developmental stages, they might even be able to figure out what babies ate versus adult fare, for instance.

In a review paper published in *Science* last October, Peter S. Ungar of the University of Arkansas and Matt Sponheimer of the University of Colorado at Boulder observed that recent analyses have hinted at unexpected diversity and complexity in the diets of our predecessors. Whereas *Ardipithecus ramidus*, one of the earliest putative hominins, dined primarily on C3 foods, as savanna chimpanzees do, other early African hominins appear to have eaten a mix of C3 and C4 foods. One species, *Paranthropus robustus*, even ate a mostly C4 diet, as Thure Cerling of the University of Utah and his colleagues reported last June in the *Proceedings of the National Academy of Sciences USA*. Scientists will no doubt be eager to see where on the dietary spectrum *A. sediba* falls and how that picture fits with emerging clues about the paleoenvironment at Malapa, which appears to have included an abundance of grasses and trees. Perhaps the dietary evidence will shine a light on how *A. sediba* was using that dexterous hand, with its apparent adaptations to tool use—and, by the same token, whether it used its long, apelike arms to forage in the trees.

END OF DAYS

THE FINAL DAYS of the Malapa hominins appear to have been grim ones. Possible drought conditions may have made water hard to come by. Berger suspects that the hominins, desperate for a drink, may have tried to climb down into the then 30- to 50-meter-deep underground cavern at Malapa to access a shallow pool of freshwater and, in so doing, tumbled to their deaths. Perhaps the boy fell in first, and the adult female—maybe his mother—tried to rescue him only to fall in herself. A menagerie of other beasts, from antelopes to zebras, met the same fate, becoming entombed alongside the hominins for posterity.

Intriguingly, geologic evidence from the site indicates that the fossil assemblage at Malapa formed right around the same time that the earth was undergoing a geomagnetic reversal—a mysterious event in which the planet's polarity flips and magnetic north becomes magnetic south. The timing raises the question of whether the reversal somehow played a role in the demise of these creatures.

Scientists know very little about why reversals occur and whether they precipitate environmental change. Some geologists have suggested that these events could conceivably wreak ecological havoc—by compromising the magnetic field that shields organisms from deadly radiation, for example, or by confusing the internal navigation systems of migratory birds and other animals that use the earth's magnetic field to orient themselves. As one of the only places in the world that has a terrestrial record of a reversal and a collection of fossils from

the same time, Malapa could offer rare insights into what happens when the planet's poles trade places.

Other evidence might throw additional light on their deaths. The fossilized bones of a pregnant antelope and her fetus from Malapa could help scientists pinpoint the time of year that the hominins died to within a couple weeks: antelopes give birth within a very narrow interval in the spring, and analysis of the fetus should allow researchers to figure out how far along the antelope was before she died. Meanwhile traces of maggots and carrion beetles that set on the hominins after death could reveal how long the bodies were exposed before the cave's flowing sediments buried them.

In a sense, the work on *A. sediba* has only just begun. "You're walking all over hominin fossils," Berger tells visitors to Malapa on an austral spring morning in late November. They are standing on the rocky ground between the tree where Matthew found the clavicle and the mining pit where Berger found its owner. Climbing down into the pit, he points onlookers to bits of fossils peeking out of the rock and awaiting collection. The awestruck guests crane to glimpse an infant's arm bone, the lower jaw of a false saber-toothed cat, the area that appears to contain the rest of the young male's skeleton. Just by gathering remains exposed by the miners and the occasional rainstorm, the team has amassed one of the largest fossil hominin samples on record. Once the researchers begin excavating the roughly 500-square-meter site, Berger knows they will find more bones—many more. Extensive planning is under way to erect a structure to protect the site from the elements and serve as a state-of-the-art field laboratory for when they begin the formal excavation later this year, which will probe beyond the miners' leavings into the undisturbed parts of the deposit. Meanwhile, in the Malapa block lab at the University of the Witwatersrand, chunks of rock blasted from the miners' pit fill floor-to-ceiling shelves. Researchers will peer into the rocks with a CT scanner to look for hominin bones, including the adult female's missing skull.

So vast are Malapa's riches that Berger could probably spend the rest of his career working on them. Yet already he is thinking about where he wants to go next. *A. sediba* "has taught me that we really need a better record—and it's out there," he warrants. The mapping project that led Berger to Malapa identified more than three dozen new fossil sites in the Cradle alone that could potentially harbor hominin remains. He is lining up researchers to dig the most promising of those spots. Berger himself has his sights set farther afield. The Congo and Angola, among other places, have cave formations similar to the ones in the Cradle and have never been searched for hominin fossils, he observes. Perhaps there, in paleoanthropological terra incognita, he will find another unexpected emissary from the dawn of humankind that will rewrite the story of our origins once again. ■

Kate Wong is a senior editor at Scientific American.

MORE TO EXPLORE

*Australopithecus sediba: A New Species of *Homo*-like Australopithecus from South Africa.*
Lee R. Berger et al. in *Science*, Vol. 328, pages 195–204; April 9, 2010.

The September 9, 2011, issue of *Science* contains five research papers on details of *A. sediba*'s anatomy and age.

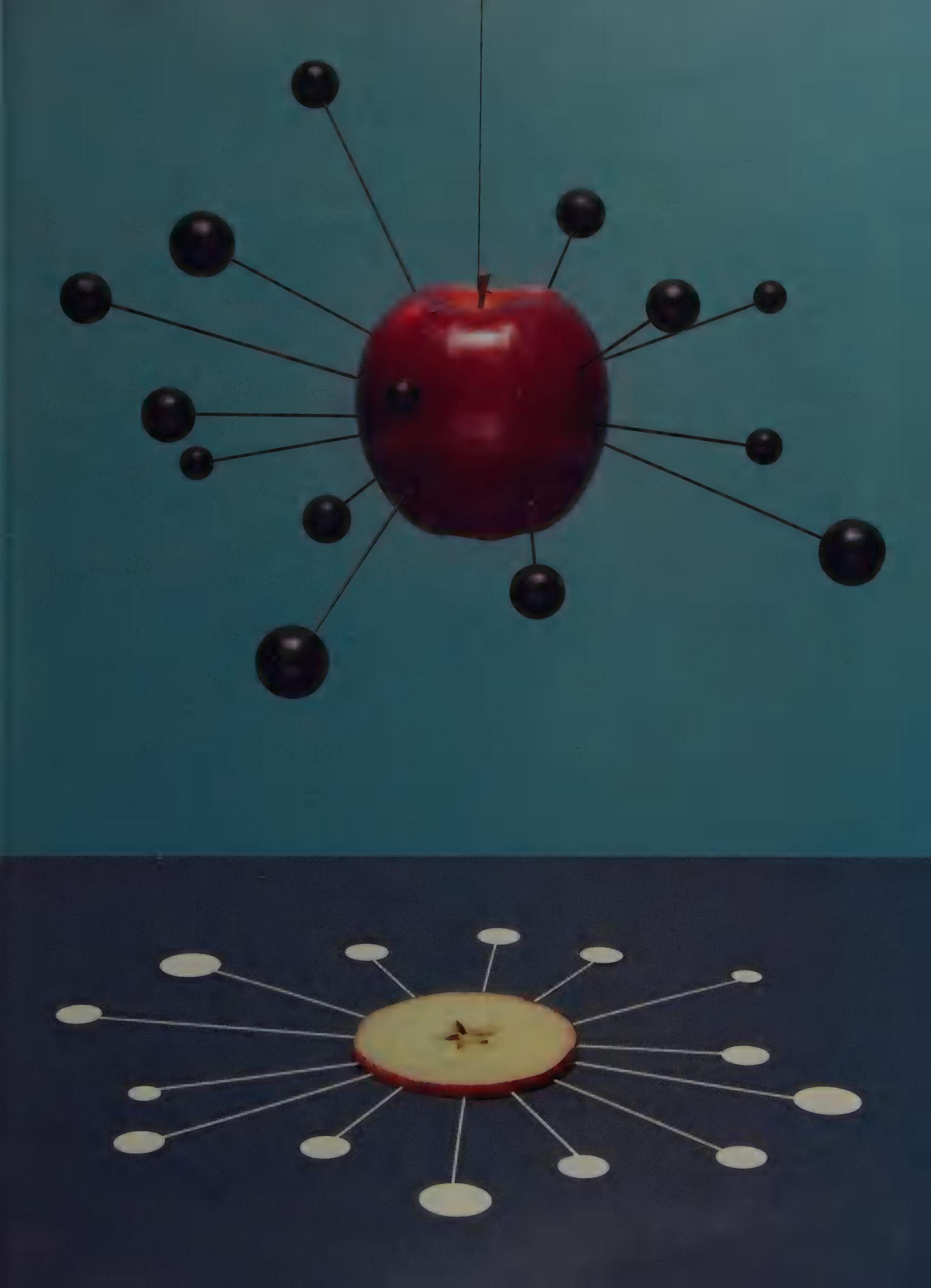
SCIENTIFIC AMERICAN ONLINE

View photographs and video of *A. sediba* at ScientificAmerican.com/apr2012/sediba

physics

Quantum Gravity in Flatland

Imagine space were 2-D rather than 3-D. How would the force of gravity work? The surprising answers are guiding physicists to a unified theory of nature. *By Steven Carlip*



Steven Carlip worked as a printer, a newspaper editor and a factory worker before deciding to become a physicist. He studied under Bryce DeWitt, one of the founding figures of quantum gravity, and is now a professor at the University of California, Davis. He is a fellow of the American Physical Society and of its British counterpart, the Institute of Physics.



From its earliest days as a science, physics has searched for unity in nature.

Isaac Newton showed that the same force responsible for the fall of an apple also holds the planets in their orbits. James Clerk Maxwell combined electricity, magnetism and light into a single theory of electromagnetism; a century later physicists added the weak nuclear force to form a unified “electroweak” theory. Albert Einstein joined space and time themselves into a single spacetime continuum.

Today the biggest missing link in this quest is the unification of gravity and quantum mechanics. Einstein’s theory of gravity, his general theory of relativity, describes the birth of the universe, the orbits of planets and the fall of Newton’s apple. Quantum mechanics describes atoms and molecules, electrons and quarks, the fundamental subatomic forces, and much besides. Yet in the places where both theories should apply—where both gravity and quantum effects are strong, such as black holes—they also seem incompatible. Physicists’ best efforts to combine them into a quantum theory of gravity have failed miserably, giving answers that make no sense or no answers at all. Despite 80 years of work by generations of physicists, including a dozen or so Nobel laureates, a quantum theory of gravity remains elusive.

Ask a physicist too hard a question, and a common reply will be, “Ask me something easier.” Physics moves forward by looking at simple models that capture pieces of a complex reality. Researchers have worked on numerous such models for quantum gravity, including approximations that apply when gravity is weak or in special cases such as black holes. Perhaps the most unusual approach is to neglect a whole dimension of space and work out how gravity would operate if our universe were only two-dimensional. (Technically, physicists refer to this situation

as “(2+1)-dimensional,” meaning two dimensions of space plus one of time.) The principles that govern gravity in this simplified universe might also apply to our 3-D one, thus giving us some much needed clues to unification.

The idea of dropping down a dimension has a distinguished history. Edwin Abbott’s 1884 novel *Flatland: A Romance of Many Dimensions* follows the adventures of “A Square,” a resident of a 2-D world of triangles, squares and other geometric figures. Although Abbott intended it as a satirical commentary on Victorian society—*Flatland* had a rigid class hierarchy, with linear women at the bottom and a class of circular priests at the top—*Flatland* also triggered a surge of interest in geometry in diverse dimensions and remains popular today among mathematicians and physicists. Researchers trying to wrap their minds around a higher-dimensional realm start by imagining what our 3-D world would look like to A Square [see “Mathematical Games,” by Martin Gardner; SCIENTIFIC AMERICAN, July 1980]. Flatland has also inspired physicists studying materials such as graphene that really do behave like 2-D spaces [see “Carbon Wonderland,” by Andre K. Geim and Philip Kim; SCIENTIFIC AMERICAN, April 2008].

The first studies of Flatland gravity, made in the early 1960s,

IN BRIEF

Stymied by the difficulty of unifying quantum mechanics with Einstein’s general theory of relativity, physicists have turned to a simplified version of the problem: imagining space to be just two-dimensional and asking how gravity would then operate.

At first, they expected 2-D gravity to be trivial. Shoehorned into one fewer dimension, gravity would become so tightly circumscribed that gravitational waves could not propagate, in which case quantum gravity should be a nonstarter.

Physicists have found it is not so trivial after all. Waves might not ripple through the continuum, but the universe as a whole could morph. The resulting quantum theory of gravity solves various puzzles of unification, such as how time may emerge from timeless physics.

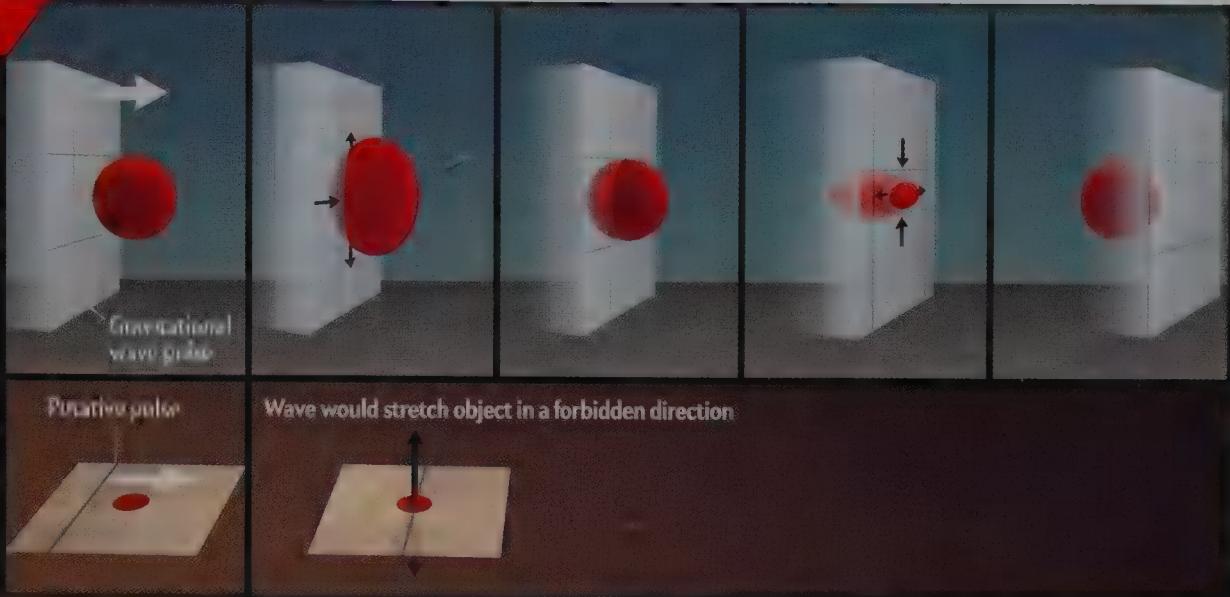
How Gravity Works in 2-D

If you took 3-D space and flattened it to 2-D, matter would not just be a lot thinner. The force of gravity would behave in fundamentally different ways. Imagining gravity in 2-D has given

physicists some helpful practice for how to merge Einstein's theory of gravity (the general theory of relativity) with quantum mechanics to create a quantum theory of gravity.

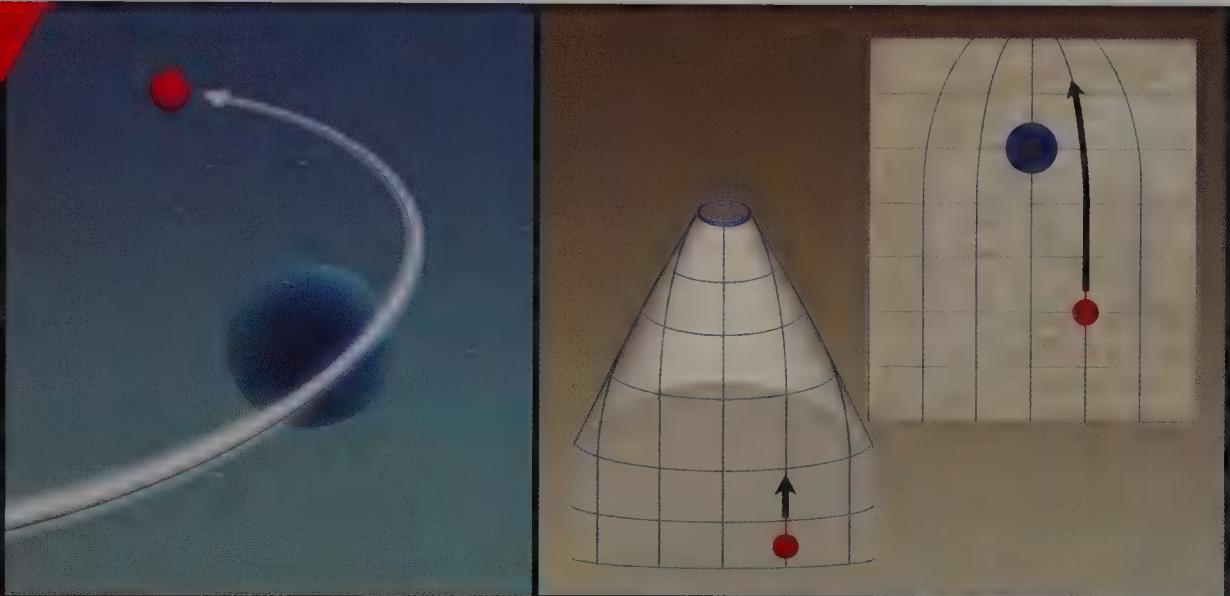
Waves Are Stillborn

According to general relativity, changes in the gravitational field propagate through space as gravitational waves, which are irreducibly 3-D; they propagate in one direction and rhythmically stretch objects in two perpendicular directions (top panel). They are unable to propagate in 2-D (bottom). Without waves, physicists are left at a loss for how to quantize gravity.



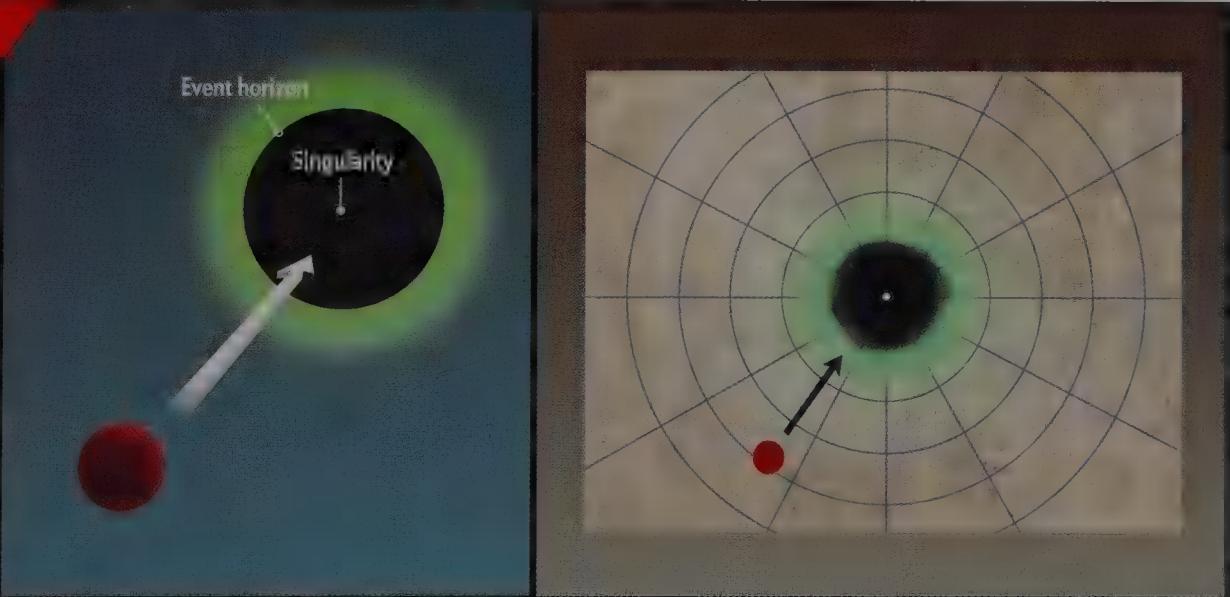
Attraction Works Differently

A massive object bends the spacetime continuum. In 3-D, this distortion causes two such objects to pull on each other according to Newton's law of universal gravitation. In 2-D, a massive object deforms space into a conical shape. Newton's law is altered: objects that move past one another are deflected onto new paths, but objects at rest remain at rest.



Black Holes Form

Gravity can go haywire under extreme conditions and give rise to phenomena not predicted by Newton's law, notably black holes—regions that objects can enter but never exit. One of the most surprising discoveries of 2-D gravity theory is that black holes can exist in 2-D space as long as the space contains dark energy. Quantum effects cause holes in both 3-D and 2-D to glow like any hot object.



were a letdown. A 2-D space literally would not have enough room for changes in the gravitational field to propagate. In the late 1980s, however, the subject had a renaissance as researchers realized that gravity works in unexpected ways. It would still sculpt the overall shape of space and even create black holes. Flatland gravity has been a case study in lateral thinking, letting us subject some of our speculative ideas, such as the so-called holographic principle and the emergence of time from timelessness, to a rigorous mathematical test.

TIME MANAGEMENT

WHEN PHYSICISTS SEEK TO DEVELOP A QUANTUM THEORY of a force, we take the corresponding classical theory as our starting point and build on it. For gravity, that means general relativity. And there the trouble starts. General relativity involves a complex system of 10 equations, each with up to thousands of terms. We cannot solve these equations in their full generality, so we face a daunting task in formulating their quantum version. But the mystery of why quantum gravity is so elusive is deeper still.

According to general relativity, the thing we call “gravity” is actually a manifestation of the shape of space and time. Earth orbits the sun not because some force tugs on it but because it is moving along the straightest possible path in a spacetime that has been warped by the sun’s mass. Uniting quantum mechanics and gravity means somehow quantizing the structure of space and time itself.

That may not sound so challenging. Yet a cornerstone of quantum mechanics is the Heisenberg uncertainty principle, the idea that physical quantities are inherently fuzzy—fluctuating randomly and having no definite values unless they are observed or undergo an equivalent process. In a quantum theory of gravity, space and time themselves fluctuate, shaking the scaffolding on which the rest of physics is built. Without a fixed spacetime as the background, we do not know how to describe positions, rates of change or any of the other basic quantities of physics. Simply put, we do not know what a quantum spacetime means.

These general obstacles to conceptualizing quantized spacetime show up in several specific ways. One is the notorious “problem of time.” Time is fundamental to our observed reality. Almost every theory of physics is ultimately a description of the way some piece of the universe changes in time. So we physicists had better know what “time” means, and the embarrassing truth is that we do not.

To Newton, time was absolute—standing outside nature, affecting matter but unaffected by it. The usual formulations of quantum mechanics accept this idea of an absolute time. Relativity, however, dethroned absolute time. Different observers in relative motion disagree about the passage of time and even about whether two events are simultaneous. A clock—as well as anything else that varies in time—runs more slowly in a strong gravitational field. No longer merely an external parameter, time is now an active participant in the universe. But if there is no ideal clock sitting outside the universe and determining the pace of change, the passage of time must arise from the internal structure of the universe [see “Is Time an Illusion?” by Craig Callender; SCIENTIFIC AMERICAN, June 2010]. But how? It is hard to even know where to start.

The problem of time has a less famous cousin, the problem of observables. Physics is an empirical science; a theory must make

The entire universe is described by a single quantum wave function, which includes all time, past, present and future. But how does it give rise to the dynamic world we live in?

verifiable predictions for observable quantities. In ordinary physics, these quantities are ascribed to specific locations: the strength of the electric field “here” or the probability of finding an electron “there.” We label “here” or “there” with the coordinates x , y and z , and our theories predict how observables depend on the values of these coordinates.

Yet according to Einstein, spatial coordinates are arbitrary, human-made labels, and in the end the universe does not care about them. If you cannot identify a point in spacetime objectively, then you cannot claim to know what is going on at it. Charles

Torre of Utah State University

has shown that a quantum theory of gravity can have no purely local observables—that is, observables whose values depend on only a single point in spacetime. So scientists are left with nonlocal observables, quantities whose values depend on many points at once. In general, we do not even know how to define such objects, much less use them to describe the world we observe.

A third problem is how the universe came into being. Did it pop into existence from nothing? Did it split off a parent universe? Or did it do something else entirely? Each possibility poses some difficulty for a quantum theory of gravity. A related problem is a perennial favorite of science-fiction writers: wormholes, which form shortcuts between locations in space or even in time. Physicists have thought seriously about this idea—in the past 20 years they have written more than 1,000 journal articles on wormholes—without settling the question whether such structures are possible.

A final set of questions revolves around the most mysterious beasts known to science: black holes. They may offer our best window into the ultimate nature of space and time. In the early 1970s Stephen Hawking showed that black holes should glow like a hot coal—emitting radiation with a so-called blackbody spectrum. In every other physical system, temperature reflects the underlying behavior of microscopic constituents. When we say a room is hot, what we really mean is that the molecules of air inside it are moving energetically. For a black hole the “molecules” must be quantum-gravitational. They are not literally molecules but some unknown microscopic substructure—what a physicist would call “degrees of freedom”—that must be capable of changing. No one knows what they truly are.

AN UNATTRACTIVE MODEL

AT FIRST GLANCE, Flatland seems an unpromising place to seek answers to these questions. Abbott’s Flatland had many laws, but a law of gravity was not among them. In 1963 Polish physicist Andrzej Staruszkiewicz worked out what that law might be by applying general relativity. He found that a massive object in Flatland would bend the 2-D space around it into a cone, like a party hat made by twisting a flat piece of paper. A small object passing the apex of this cone would find its path deflected, much

as the sun bends a comet's path in our universe. In 1984 Stanley Deser of Brandeis University, Roman Jackiw of the Massachusetts Institute of Technology and Gerard 't Hooft of Utrecht University in the Netherlands worked out how quantum particles would move through such a space.

This geometry would be much simpler than the complicated pattern of curvature that gravity causes in our 3-D universe. Flatland would lack the equivalent of Newton's law of attraction; instead the strength of the force would depend on objects' velocities, and two bodies at rest would not be pulled toward each other. This simplicity is appealing. It suggests that quantizing Staruszkiewicz's theory would be easier than quantizing full-blown general relativity in 3-D. Unfortunately, the theory is too simple: nothing is left to quantize. A 2-D space has no room for an important element of Einstein's theory: gravitational waves.

Consider the simpler case of electromagnetism. Electric and magnetic fields are produced by electric charges and currents. As Maxwell showed, these fields can detach themselves from their sources and move freely as light waves. In the quantum version of Maxwell's theory, the waves become photons, the quanta of light. In the same way, the gravitational fields of general relativity can detach themselves from their sources and become freely propagating gravitational waves, and physicists widely assume that a quantum theory of gravity will contain particles called gravitons that do the traveling.

A light wave has a polarization: its electric field oscillates in a direction perpendicular to its direction of motion. A gravitational wave also has a polarization, but the pattern is more complicated: the field oscillates not in one but in two directions perpendicular to its direction of motion [see box on page 43]. Flatland has no room for this behavior. Once the direction of motion

is fixed, only one perpendicular direction remains. Gravitational waves and their quantum counterparts, gravitons, simply cannot be squeezed into two dimensions of space.

Despite occasional sparks of interest, Staruszkiewicz's discovery languished. Then, in 1989, Edward Witten of the Institute for Advanced Study in Princeton, N.J., stepped in. Witten, widely considered the world's leading mathematical physicist, had been working on a special class of fields in which waves do not propagate freely. When he realized that 2-D gravity fit into this class, he added the crucial missing ingredient: topology.

DOUGHNUTLAND

WHAT WITTEN POINTED OUT was that even if gravity cannot propagate as waves, it can still have a dramatic effect on the overall shape of space. This effect does not arise when Flatland is just a plane; it requires a more complicated topology. When an ice sculpture melts away, the details become muted, but certain features such as holes tend to last. Topology describes these features. Two surfaces have the same topology if one can be smoothly deformed into the other without cutting, tearing or gluing. For instance, a hemisphere and a disk share the same topology: stretching the hemisphere by pulling on its perimeter yields a disk. A sphere has another topology: to turn it into a hemisphere or disk, you would need to snip out a piece. A torus, like the surface of a doughnut, has yet another. The surface of a coffee cup has the same topology as a torus: the handle looks like a torus, and the rest of the cup can be smoothed out without cutting or tearing—thus, the old mathematician's joke that a topologist cannot tell a doughnut from a coffee cup.

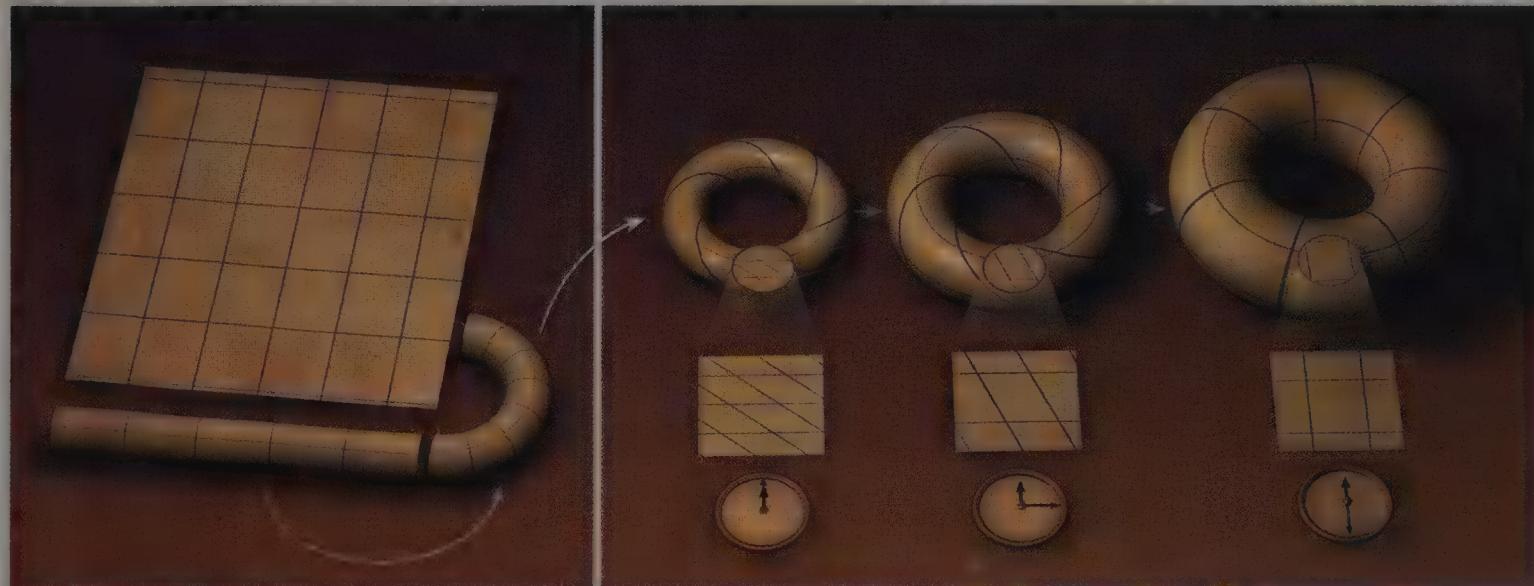
Although tori look curved, when you consider their internal geometry rather than their shape as seen from the outside, they

QUANTUM GRAVITY AS A HOLISTIC EFFECT

How to Quantize Gravity in 2-D

Two-dimensional gravity has given physicists a new perspective on what gravity is. It is not necessarily a force that propagates through space—indeed, in two dimensions it cannot propagate at all. Instead gravity can also be the driver of changes in the overall shape of space. Physicists have studied a

square or parallelogram universe that has been rolled into a torus. Tori of different sizes and shapes correspond to the 2-D universe at different moments in time. What happens in any small region of space mirrors the general condition of space; microcosm and macrocosm are inextricably linked.



can actually be flat. What makes a torus a torus is the fact you can make a full loop around it in two separate directions: through the hole or around the rim. This feature will be familiar to anyone who has played a 1980s-era video game where a combatant exiting the right side of the screen reenters on the left. The screen is flat: it obeys the rule of plane geometry, such as the fact that parallel lines never meet. Yet the topology is toroidal.

In fact, an infinite family of such tori exist—all flat but all distinct, labeled by a parameter called the modulus. What gravity in a toroidal universe does is to cause the modulus to evolve in time. A torus starts as a line at the big bang and opens up to assume an ever more square-shaped geometry as the universe expands [see box on preceding page]. Starting with Witten's results, I showed that this process could be quantized, turning the classical theory of gravity into a quantum one. Quantum gravity in Flatland is a theory not of gravitons but of shape-shifting tori. That view marks a shift in the usual picture of quantum theory as a theory of the very small. Quantum gravity in two dimensions is, in fact, a theory of the entire universe as a single object. This insight gives us a rich enough model to explore some of the fundamental conceptual problems of quantum gravity.

FINDING THE TIME

FLATLAND GRAVITY DEMONSTRATES, for example, how time might emerge from a fundamentally timeless reality. In one formulation of the theory, the entire universe is described by a single quantum wave function, similar to the mathematical device that physicists routinely use to describe particles and atoms. This wave function does not depend on time, because it already includes all time, past, present and future, in one package. Somehow this “timeless” wave function gives rise to the change we observe in the world. The trick is to remember Einstein's aphorism that time is what is measured by a clock. Time does not stand outside the universe; it is determined by a subsystem that is correlated with the rest of the universe, just as a wall clock is correlated with Earth's rotation.

The theory offers many different clock options, and our choice

defines what we mean by “time.” A Square can define time by using the readings of atomic clocks in satellites, like those in the GPS. He can label time by the lengths of curves extending from the big bang, by the size of his expanding universe, or by the amount of redshift caused by its expansion. Once he has made such a choice, all other physical observables change with clock time. The modulus of the torus universe is correlated with its size, for instance, and A Square perceives this as a universe evolving in time. The theory thus bootstraps time from a timeless universe. These ideas are not new, but quantum gravity in Doughnutland has at last given us a setting in which we can do the math and check that the picture does not just look pretty but really works. Some of the definitions of time have intriguing consequences, such as implying that space can be creased.

As for the problem of observables, Doughnutland gives us a set of objectively measurable quantities—namely, the moduli. The twist is that these quantities are nonlocal: they do not reside at specific locations but describe the structure of the whole space. Anything that A Square measures is ultimately a proxy for these nonlocal quantities. In 2008 Catherine Meusburger, now at the University of Erlangen-Nürnberg in Germany, showed how these moduli relate to real cosmological measurements such as time delays and redshifts for beams of light. I have shown how they relate to objects' motion.

Flatland gravity offers good news for fans of wormholes: at least one formulation of the theory permits the topology of space to change. A Square could go to bed tonight in Sphereland and wake up tomorrow in Doughnutland, which is equivalent to creating a shortcut between two distant corners of the universe. In some versions of the theory, we can describe the creation of the universe out of nothing, the ultimate change in topology.

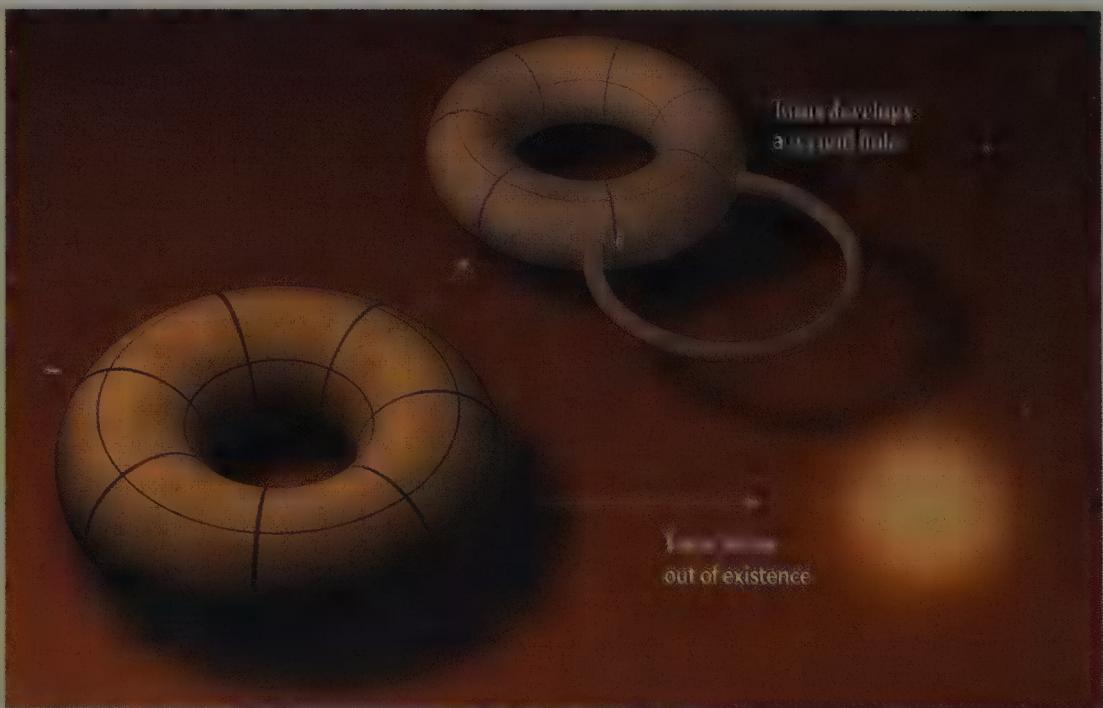
ON THE EDGE OF SPACE

BECAUSE GRAVITY IN FLATLAND IS STUNTED, it used to be common knowledge among experts in the field (me included) that 2-D black holes were impossible. In 1992, though, three physicists—Máximo Bañados, now at the Pontifical Catholic University of

CHANGING TOPOLOGY

Wormholes and Bangs

In a quantum theory of gravity, unlike Einstein's theory, the topology of the universe can change, which might solve some long-standing questions about the universe. For instance, a one-holed torus could become a two-holed one, which would amount to creating a wormhole—a backdoor passage from one location to another. Wormholes might conceivably be used as time machines. Also, the cosmos could pop out of existence or be born from sheer nothingness.



Chile in Santiago, and Claudio Bunster (then Claudio Teitelboim) and Jorge Zanelli, both at the Center for Scientific Studies in Valdivia, Chile—shocked the world, or at least our little corner of it, by showing that the theory does allow black holes, as long as the universe has a certain type of dark energy.

A so-called BTZ black hole is very much like a real black hole in our own universe. Formed from matter collapsing under its own weight, it is surrounded by an event horizon, a one-way barrier from which nothing can escape. To an observer who remains on the outside, the event horizon looks like an edge of the universe: any object that falls through the horizon is completely cut off from us. Per Hawking's calculations, A Square should see it glow at a temperature that depends on its mass and spin.

That result presents a puzzle. Lacking gravitational waves or gravitons, Flatland gravity should also lack the gravitational degrees of freedom that would explain black hole temperature. Yet they sneak in anyway. The reason is that the event horizon itself provides some additional structure that empty 2-D space lacks. The horizon exists at a certain location, which, mathematically, augments the raw theory with some additional quantities. Vibrations that wiggle the horizon provide degrees of freedom. Remarkably, we find that they exactly reproduce Hawking's results.

Because the degrees of freedom are features of the horizon, they reside, in a sense, on the edge of Flatland itself. So they are a concrete realization of a fascinating proposal about the nature of quantum gravity, the holographic principle. This principle suggests that dimension may be a fungible concept. Just as a hologram captures a three-dimensional image on a flat 2-D film, many physicists speculate that the physics of a d -dimensional world can be completely captured by a simpler theory in $d-1$ dimensions. In string theory—a leading effort to unify general relativity and quantum mechanics—this idea led in the late 1990s to a novel approach for creating a quantum theory of gravity [see “The Illusion of Gravity,” by Juan Maldacena; SCIENTIFIC AMERICAN, November 2005].

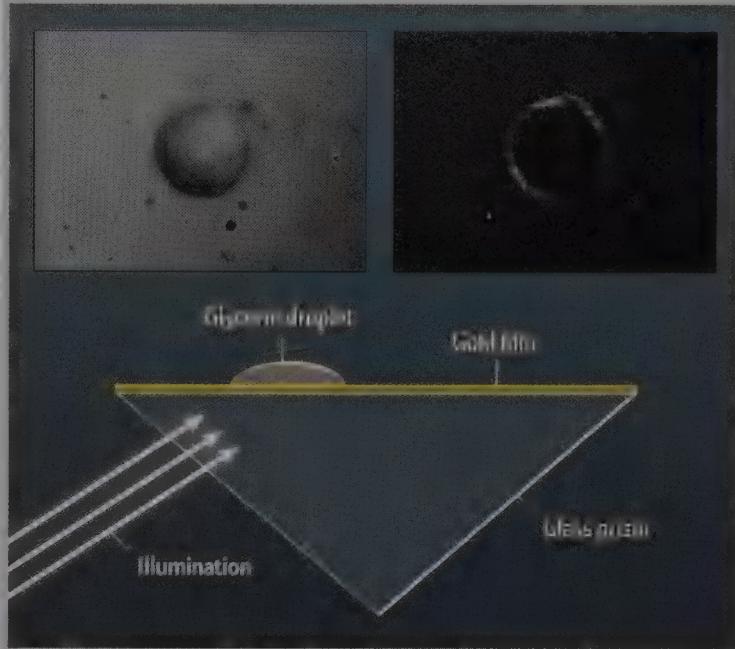
Flatland gravity provides a simplified scenario to test that approach. Just over four years ago Witten and Alexander Maloney, now at McGill University, again surprised the physics world by suggesting that the holographic predictions appear to fail for the simplest form of 2-D gravity. The theory, they found, seemed to predict impossible thermal properties for black holes. This unexpected result suggests that gravity is an even more subtle phenomenon than we had suspected, and the response has been a fresh surge of Flatland research. Perhaps gravity simply does not make sense by itself but must work in partnership with other kinds of forces and particles. Perhaps Einstein's theory needs to be revised. Perhaps we need to find a way to put back some local degrees of freedom. Perhaps the holographic principle does not always hold. Perhaps space, like time, is not a fundamental ingredient of the universe. Whatever the answer, Flatland gravity has pointed us in a direction we might not otherwise have taken.

Although we cannot make a real 2-D black hole, we might be able to test some of the predictions of the Flatland model experimentally. Several laboratories around the world are working on 2-D analogues of black holes. For example, a fluid flowing faster than the speed of sound produces a sonic event horizon from which sound waves cannot escape. Experimenters have also built 2-D black holes using electromagnetic waves confined to surface

EXPERIMENTAL ANALOGUES

Flatland for Real

A laboratory system that mimics Flatland, developed by Igor I. Smolyaninov of the University of Maryland and his colleagues, is a metal surface along which electromagnetic waves propagate. These 2-D analogues of light are known as surface plasmons. A liquid droplet traps them much as a 3-D black hole traps photons; the analogue of the event horizon shows up as a white rim (below right). Just as theorists find Flatland gravity a useful warm-up exercise for unifying physics, experimentalists think 2-D systems will have practical applications in optics.



es [see box above]. Such analogues should also exhibit a quantum glow in much the same way a black hole does [see “Hawking Was Right (Probably),” by John Matson; SCIENTIFIC AMERICAN, December 2010].

Quantum gravity in Flatland began as a playground for physicists, a simple setting in which to explore ideas about real-world quantum gravity. It has already taught us valuable lessons about time, observables and topology that are carrying over to real 3-D gravity. The model has surprised us with its richness: the unexpectedly important role of topology, its remarkable black holes, its strange holographic properties. Perhaps soon we will fully understand what it is like to be a square living in a flat world. ■

MORE TO EXPLORE

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Steven Carlip's Web page offers a glossary of quantum gravity: http://snurl.com/carlip_lctoc.html

SCIENTIFIC AMERICAN ONLINE

If the dimensional machinations of gravity have left your head spinning, watch an explanatory video at ScientificAmerican.com/apr2012/quantum-gravity

THIS IS YOUR BRAIN

Neural circuits responsible for conscious self-control are highly vulnerable to even mild stress. When they shut down, primal impulses go unchecked and mental paralysis sets in

By Amy Arnsten, Carolyn M. Mazure and Rajita Sinha



IN MELTDOWN

TD

HE ENTRANCE EXAM TO MEDICAL SCHOOL consists of a five-hour fusillade of hundreds of questions that, even with the best preparation, often leaves the test taker discombobulated and anxious. For some would-be physicians, the relentless pressure causes their reasoning abilities to slow and even shut down entirely. The experience—known variously as choking, brain freeze, nerves, jitters, folding, blanking out, the yips or a dozen other descriptive terms—is all too familiar to virtually anyone who has flubbed a speech, bumped up against writer's block or struggled through a lengthy exam.

For decades scientists thought they understood what happens in the brain during testing or a battlefield firefight. In recent years a different line of research has put the physiology of stress in an entirely new perspective. The response to stress is not just a primal reaction affecting parts of the brain that are common to a wide array of species ranging from salamanders to humans. Stress, in fact, can cripple our most advanced mental faculties, the areas of the brain most developed in primates.

Older textbooks explained that the hypothalamus, an evolutionarily ancient structure lodged at the base of the brain, reacts to stress by triggering the secretion of a wave of hormones from the pituitary and adrenal glands, which makes the heart race, elevates blood pressure and diminishes appetite. Now research reveals an unexpected role for the prefrontal cortex, the

Amy Arnsten is a professor of neurobiology at the Yale School of Medicine. Her research on molecular changes in the prefrontal cortex during stress and aging has led to treatments such as prazosin and guanfacine for post-traumatic stress disorder, attention-deficit hyperactivity disorder and other conditions.

Carolyn M. Mazure is a professor of psychiatry and psychology and associate dean for faculty affairs at the Yale School of Medicine. She created and directs Yale's interdisciplinary women's health research center.

Rajita Sinha directs the Yale Stress Center, which focuses on understanding the effects of stress on behavior. She is a professor of psychiatry at the Yale School of Medicine.



area immediately behind the forehead that serves as the control center that mediates our highest cognitive abilities—among them concentration, planning, decision making, insight, judgment and the ability to retrieve memories. The prefrontal cortex is the part of the brain that evolved most recently, and it can be exquisitely sensitive to even tempo-

rary everyday anxieties and worries.

When things are going well, the prefrontal cortex acts as a control center that keeps our baser emotions and impulses in check. The new research demonstrates that acute, uncontrollable stress sets off a series of chemical events that weaken the influence of the prefrontal cortex while strengthening the dominance of older parts of the brain. In essence, it transfers high-level control over thought and emotion from the prefrontal cortex to the hypothalamus and other earlier evolved structures. As the older parts take over, we may find ourselves either consumed by paralyzing anxiety or else subject to impulses that we usually manage to keep in check: indulgence in excesses of food, drink, drugs or a spending spree at a local specialty store. Quite simply, we lose it.

The growing understanding that acute stress can severely

IN BRIEF

Freezing under stress, a common experience for all of us at some point in our life, has its roots in a loss of control over "executive functions" that allow us to control our emotions.

Prefrontal cortical areas, which serve as the brain's executive command centers, normally hold our emotions in check by sending signals to tone down activity in primitive brain systems.

Under even everyday stresses, the prefrontal cortex can shut down, allowing the amygdala, a locus for regulating emotional activity, to take over, inducing mental paralysis and panic.

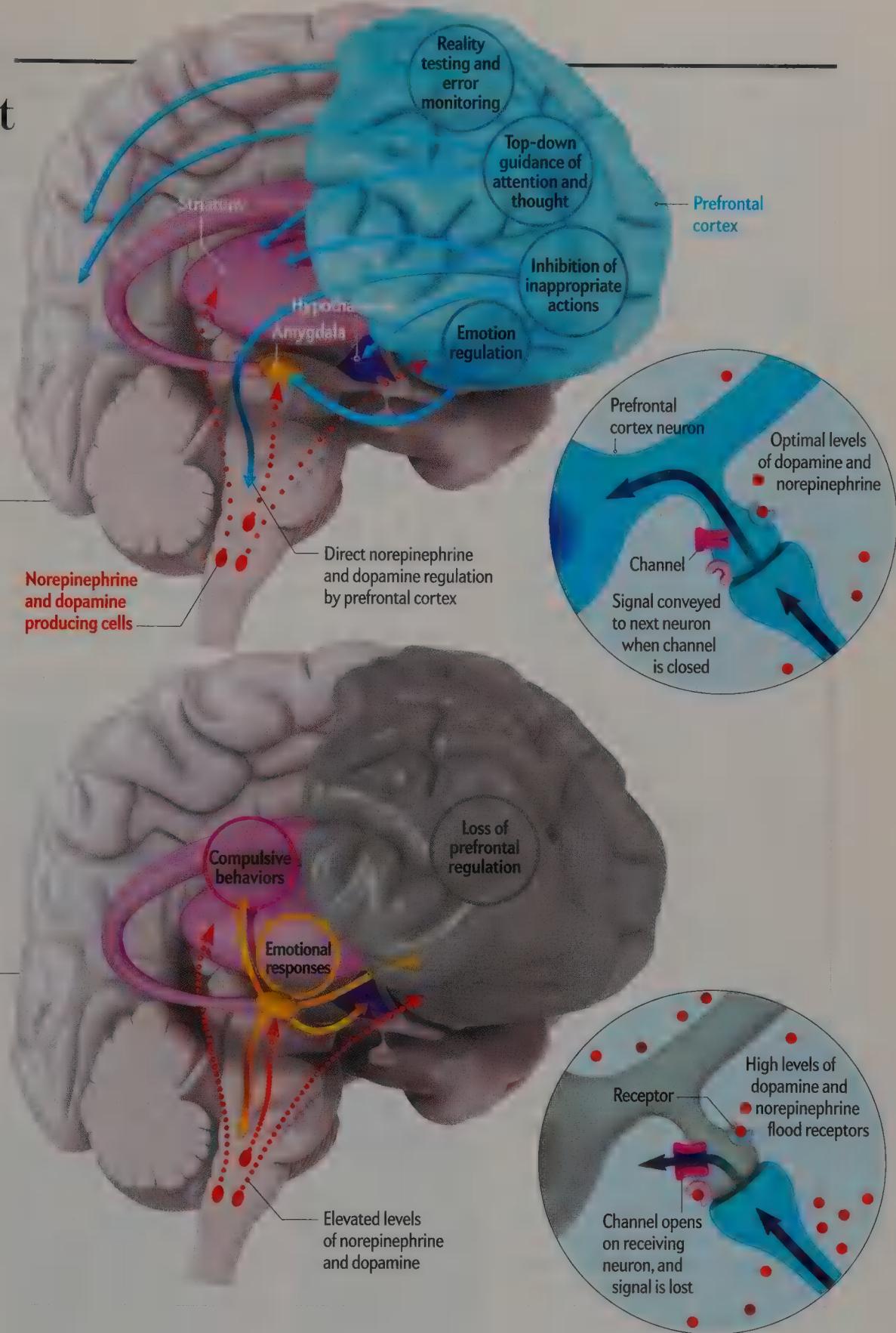
Researchers are probing further the physiology of acute stress and are considering behavioral and pharmaceutical interventions to help us retain composure when the going gets tough.

How We Lose It

The area just behind your forehead is the brain's executive control center. The prefrontal cortex, as it is known, is responsible for our ability to inhibit inappropriate impulses. Ordinary, everyday acute stresses are capable, however, of undermining this basic sense of self-control, allowing emotionality and impulsivity to take over.

Unstressed

Signals from the prefrontal cortex move to areas deep within the brain to regulate our habits (striatum), basic appetites such as hunger, sex and aggression (hypothalamus), and emotional responses such as fear (amygdala). The prefrontal cortex also regulates the stress responses from the brain stem, including the activity of neurons that make norepinephrine and dopamine. Moderate levels of these two neurotransmitters engage receptors that strengthen connections to the prefrontal cortex (inset).



compromise the function of higher "executive" areas in the human brain has drawn the interest of investigators. They are now not just trying to understand what happens in your head when you freeze but also developing behavioral and pharmaceutical interventions to help you keep your composure.

MIND THE JITTERS

WHY WE LOSE IT has fascinated scientists for decades. After World War II, investigators analyzed why pilots who were highly skilled in peacetime made simple but fatal mistakes in maneuvering their craft during the heat of battle. What actually

happens behind the human skull's frontal bone remained a mystery until the relatively recent arrival of neuroimaging techniques. In a brain scanner, the riot of activity in the prefrontal cortex gives a clue to just how vulnerable the brain's master controller is.

The prefrontal cortex is so sensitive to stress because of its special status within the hierarchy of brain structures. It is the most highly evolved brain region, bigger proportionally in humans than in other primates, and makes up a full third of the human cortex. It matures more slowly than any other brain area and reaches full maturity only after the teen years have

passed. The prefrontal area houses the neural circuitry for abstract thought and allows us to concentrate and stay on task, while storing information in the mental sketch pad of working memory. This temporary memory storage area operates by allowing us to keep “in mind” such information as the sum of digits that need to be carried over to the next column when performing addition. As a mental-control unit, the prefrontal area also inhibits inappropriate thoughts and actions.

The neurological executive center functions through an extensive internal network of connections among the triangular-shaped neurons called pyramidal cells. These neurons also send out connections to more distant reaches of the brain that control our emotions, desires and habits. When unstressed, the circuits in this network hum along contentedly. Working memory reminds us to start that assignment due next week, and other circuitry sends a message to lower brain regions signaling that it is perhaps best to forgo a second glass of wine. Meanwhile a message to the amygdala, a deep-brain structure that controls fear reactions, provides assurance that the huge hulk approaching on the sidewalk is not about to smash you in the face.

Keeping this network firing as it should can be a fragile process—and when stress hits, even small changes in the neurochemical environment can instantly weaken network connections. In response to stress, our brain floods with arousal chemicals such as norepinephrine and dopamine, which are released by neurons in the brain stem that send projections throughout the brain. Elevated levels of these signaling chemicals in the prefrontal cortex shut off neuron firing, in part by weakening the connection points, or synapses, between neurons temporarily. Network activity diminishes, as does the ability to regulate behavior. These effects only worsen as the adrenal glands near the kidneys, on command from the hypothalamus, spritz the stress hormone cortisol into the bloodstream, sending it to the brain. In this circumstance, self-control depends on a tricky balancing act.

“Keeping one’s cool” is an expression that accurately represents a description of the underlying biological processes. The neural machinery of the prefrontal cortex—and its ability to muster working memory to stay focused

on the task at hand—may keep the cascade of neurotransmitters generated deep within the brain from triggering a panicked tide of emotion.

Our research clarifying how easily the prefrontal cortex can be shut down started about 20 years ago. Studies in animals by one of us (Arnsten), along with the late Patricia Goldman-Rakic of Yale University, were among the first to illustrate how neurochemical changes during stress can rapidly switch off prefrontal function. The work showed that neurons in the prefrontal cortex disconnect and stop firing after being exposed to a flood of neurotransmitters or stress hormones.

In contrast, areas deep within the brain take a stronger hold over our behavior. Dopamine arrives at a series of deep-brain structures, collectively called the basal ganglia, that regulate cravings and habitual emotional and motor responses. The basal ganglia hold sway not only when we ride a bicycle without falling but also when we indulge in addictive habits, such as those that make us long for that forbidden ice cream.

In 2001 Benno Roozendaal, now at the University of Groningen in the Netherlands, James McGaugh of the University of California, Irvine, and their colleagues found similar changes in the amygdala, another older brain region. In the presence of norepinephrine and cortisol, the amygdala alerts the rest of the nervous system to prepare for danger and also strengthens memories that are related to fear and other emotions.

This research now extends to humans. These studies have begun to show that some people seem more vulnerable than others because of their genetic makeup or because of a previous history of stress exposure. After dopamine and norepinephrine switch off circuits in the prefrontal area required for higher cognition, enzymes normally chew up the neurotransmitters so that the shutdown does not persist. In this way, we can return to our baseline when stress abates. Certain forms of a gene can weaken these enzymes, making people more vulnerable to stress and, in some cases, mental illness. Similarly, environmental factors can increase vulnerability; for example, lead poisoning can mimic aspects of the stress response and erode cognition.

Still other research focuses on what happens when the assault on

“KEEPING ONE’S COOL”

is an expression that accurately conveys the underlying brain physiology.



the prefrontal cortex lasts for days or weeks. Chronic stress appears to expand the intricate web of connections among neurons in our lower emotional centers, whereas the areas engaged during flexible, sustained reasoning—anything from the philosophy of Immanuel Kant to calculus—start to shrivel. Under these conditions, the branching, signal-receiving dendrites in the primal amygdala enlarge, and those in the prefrontal cortex shrink. John Morrison of the Mount Sinai School of Medicine and his colleagues have shown that prefrontal dendrites can regrow if the stress disappears, but this ability to rebound may vanish if the stress is especially severe. One of us (Sinha) has found evidence of this in humans, where the shrinkage in prefrontal gray matter relates to history of stress exposure.

This chain of molecular events makes us more vulnerable to subsequent stress and most likely contributes to depression, addiction and anxiety disorders, including post-traumatic stress. Gender appears to be a factor in determining how we react to stress. In women, the hormone estrogen may amplify sensitivity. For example, as one of us (Mazure) and her colleagues have shown, life stress poses a greater risk for depression in women than men and is more likely to reduce abstinence from certain addictive behaviors, such as smoking, for women as compared with men. In men, stress may play a more prominent role in exacerbating cravings and eliciting habitual behaviors mediated by the basal ganglia.

More work on how stress alters the brain's prefrontal self-control locus remains to be done. Some researchers are investigating how other neurochemicals affect the prefrontal cortex. Trevor W. Robbins and Angela Roberts of the University of Cambridge head one group looking at whether serotonin, which plays a key role in depression, may modulate stress and anxiety through its actions in the prefrontal cortex.

These studies remain challenging because modern ethical standards for experiments using humans require that subjects should not be exposed to situations of extreme psychological stress, and indeed human study participants are told they can stop at any time, giving them control over the experimental situation in a manner that does not mimic real-life stress. Several labs have succeeded in simulating the effects of uncontrolled stress by having study participants watch disturbing movies or, as done by the Sinha group, briefly imagine their own stressful experiences to tap into their reactions.

One question that still perplexes researchers is why the brain has built-in mechanisms to weaken its highest cognitive functions. We still do not know for sure, but the triggering of these primal reactions may perhaps have saved human lives when a predatory wild animal was lurking in the bushes. If we suddenly see a tiger burning bright in the forest, it is far more useful to freeze so that the animal cannot see us than to be remembering the words of William Blake's poem.

Absent our slow, deliberate higher-brain networks, primitive brain pathways can stop us on a dime or ready us to flee. These mechanisms may serve a similar function when we face danger in the modern world—say, when a reckless driver cuts us off and we need to slam on the brakes. If we remain in this state, though, prefrontal function weakens, a devastating handicap in circumstances where we need to engage in complex decision making about a loved one's serious medical condition or organize an important project on a tight deadline.

GET AHOOLD OF YOURSELF

A LOGICAL RESPONSE to our growing understanding of the jitters is to devise strategies to keep our neural-control center intact. Scientists hope that understanding the molecular events that cause the brain to degenerate from a "reflective" to a "reflexive" state may lead to better treatments for stress disorders. Some of these insights confirm what we already know. Training for emergencies or for military service is all about teaching the basal ganglia and other brain structures to learn the automatic reactions needed to survive. Animal research suggests that the sense of psychological control that becomes second nature to a soldier or emergency medical technician remains the deciding factor in whether we fall apart during stress. Public speaking exhilarates those who feel confident before an audience. For others, it induces nothing but terror, and their minds "go blank."

The routines of the drill sergeant are mirrored by animal studies that show that juveniles grow up to be more capable in handling stress if they have had multiple, successful experiences confronting mild stress in their youth. Similarly, human studies indicate that success in managing challenging situations can build resilience. In contrast, if children stumble through these experiences, they can become more sensitive to and burdened by stress and depression when they grow up.

Clues to new treatments may be slowly emerging from the laboratory. The drug prazosin, a generic therapy for blood pressure that blocks some of norepinephrine's detrimental actions, is being tested in veterans and civilians with post-traumatic stress disorder. Prazosin also appears to decrease both alcohol cravings and levels of consumption. A very recent study by Sherry McKee of Yale and her colleagues has found that another generic medication for blood pressure, called guanfacine, can inhibit some stress reactions and strengthen prefrontal cortical networks, helping people to resist smoking during stress exposure. (Arnsten and Yale University receive royalties from Shire Pharmaceuticals for an extended-release form of guanfacine used for treatment of attention-deficit hyperactivity disorder for children and adolescents but do not receive royalties for the immediate-release form of the drug used in adults in this study.) Further, many labs have shown that behavioral strategies such as relaxation, deep breathing and meditation can reduce the stress response.

And what about that sense of control? Perhaps by learning about how the brain reacts to stress, you may come away with an enhanced sense of control. So maybe the next time you are taking a test or speaking in public and your mind goes blank, you can say to yourself, "This is just my brain trying to save me from a tiger." Maybe it will bring a comforting smile to your face even if it does not bring the correct answer or word to mind. **SA**

MORE TO EXPLORE

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Take a simple self-assessment to test the extent you are affected by acute stress at ScientificAmerican/apr2012/stress



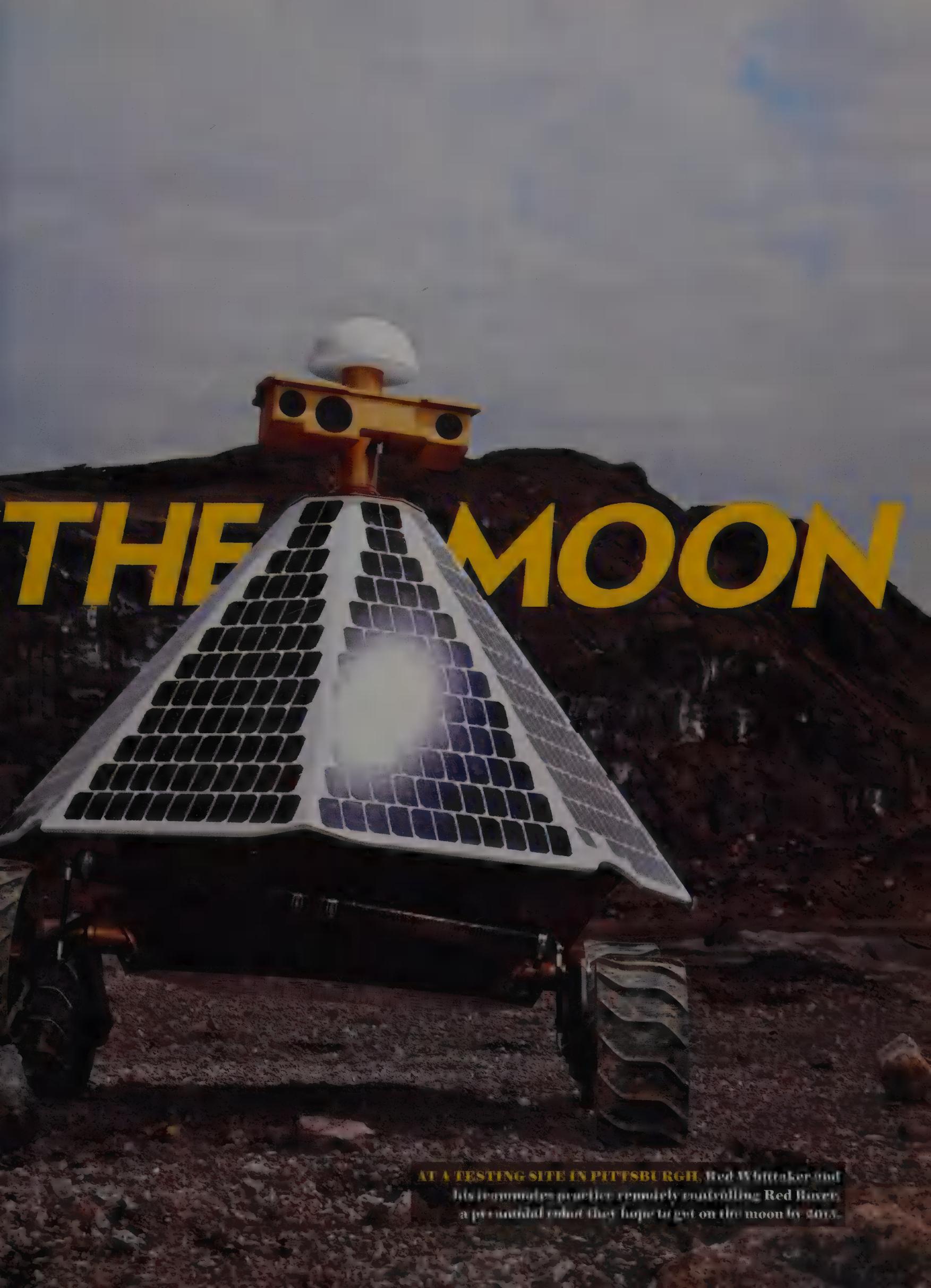
SPACE SCIENCE

BOUND FOR

The next rover to roam the moon's surface
may come not from NASA and its rocket scientists,
but from college students and
private companies working on a shoestring

By Michael Belfiore

THE MOON



AT A TESTING SITE IN PITTSBURGH, Red Whittaker and his team of engineers prepare to test Red Rover, a potential robot they hope to get to the moon by 2016.

Michael Belfiore is a freelance journalist and speaker on the innovations shaping our future. He is author of *Rocketeers: How a Visionary Band of Business Leaders, Engineers, and Pilots Is Boldly Privatizing Space* (HarperCollins, 2008).



NA MUDDY, RUBBLE-STREWN FIELD ON the banks of the Monongahela River in Pittsburgh, a five-foot-tall pyramidal robot with twin camera eyes slowly rotates on four metal wheels, its electric motors emitting a low whine. In a nearby trailer, students from Carnegie Mellon University huddle around a laptop to watch the world through the robot's eyes. In the low-resolution grayscale images on the laptop's screen, the rutted landscape looks a lot like the moon, which is the robot's ultimate destination.

Carnegie Mellon robotics professor William "Red" Whittaker and his students built Red Rover to win the Google Lunar X PRIZE, a competition designed to boost the role of private companies in space and inspire innovation in spaceflight technology. The winning prize is \$20 million, which will go to the first nongovernment team that lands a robot on the moon, gets the robot to travel half a mile or so, and sends high-definition video back to Earth—all by the end of 2015. A second-place prize of \$5 million, along with bonuses for other achievements such as reaching the site of an *Apollo* landing, brings the total purse to \$30 million. Although 26 teams are competing, Whittaker's team is a clear leader. His firm, Astrobotic Technology, was the first team to make a down payment on a rocket that will carry its spacecraft and rover to the moon. Whittaker has also proved himself to be a champion builder of autonomous vehicles that can navigate extreme environments.

The Google Lunar X PRIZE comes at a major turning point for the U.S. space program. In 2010, following the recommendations of the Review of U.S. Human Space Flight Plans Committee, President Barack Obama directed NASA to encourage privately owned and operated spaceships to replace the retiring space shuttle. With input and seed money from NASA, the reasoning goes, private companies can design and construct ships

more quickly and more affordably than the usual big contractors can produce vehicles for the government agency. In the same spirit, the Google Lunar X PRIZE seeks to foster a new class of private planetary missions, one that does not depend on expensive one-off spacecraft and political

commitments that may not last beyond one administration. Instead researchers would pay private companies to launch their rovers and instruments. NASA has added its own incentives—an additional \$30.1 million, split among six teams for surmounting technical feats that have stumped many government rovers, such as surviving the lunar night. The fate of private spaceflight companies after the Google Lunar X PRIZE is far from certain, and not everyone is convinced that a market exists for their services, but many researchers are excited about the prospect of commercially funded space science.

TEST LAUNCH

THE CONTEST has a precedent in the \$10-million Ansari X PRIZE, which ended in 2004, when SpaceShipOne became the first privately manufactured manned vehicle to leave the atmosphere. SpaceShipOne was a rocket plane built by Mojave, Calif.-based Scaled Composites, with funding from Microsoft billionaire Paul Allen. Virgin Galactic is now financing SpaceShipTwo. It has received more than \$60 million in deposits from individuals who are willing to pay \$200,000 each for the chance to float in microgravity and see Earth from a distance. NASA has contracted Virgin and six other private companies to fly scientific equipment onboard SpaceShipTwo and other spacecraft to con-

IN BRIEF

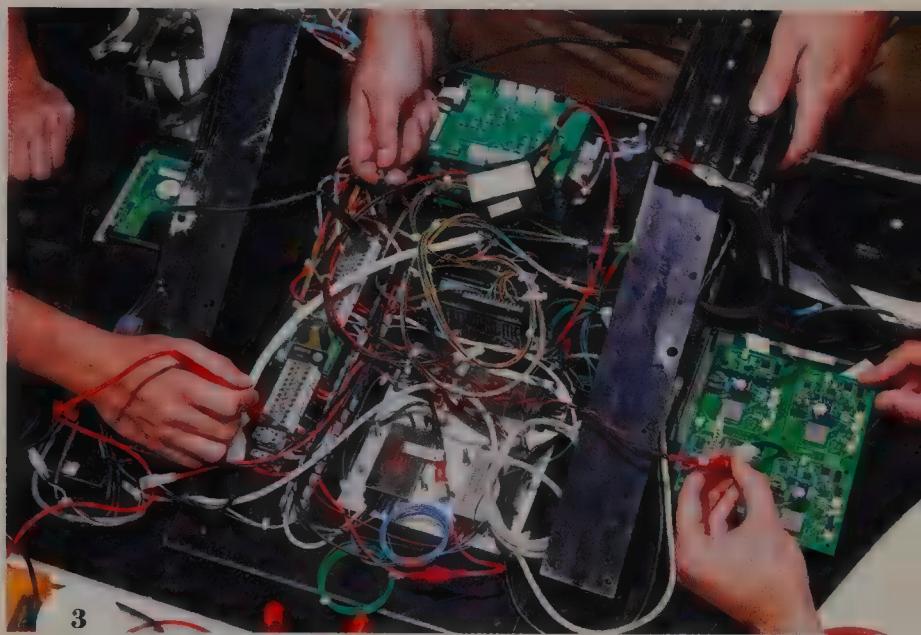
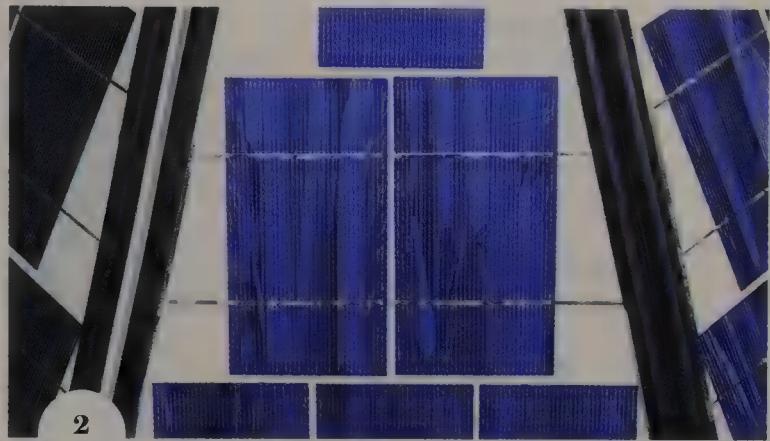
Now that NASA's space shuttle is retired, scientists may turn to privately funded rockets to get themselves and their equipment into space.

The Google Lunar X PRIZE competition offers \$20 million to the first nongovernment team to get a rover on the moon.

Of the 26 competitors, Astrobotic may stand the best chance of winning. Team leader William "Red" Whittaker has spent his career building innovative robots.



1



RED WHITTAKER, Astrobotic team leader (1), stands beside a one-tenth scale model of the Falcon 9 rocket that will launch his robot into space. Red Rover's pyramidal body is lined with solar arrays such as those shown to catch the sun's light at different angles (2). The electronics that guide Red Rover are built to survive the boiling lunar day and the freezing lunar night (3).

duct experiments on challenges such as transferring fuel without gravity. Now the organizers of the Google Lunar X PRIZE hope to duplicate this success for robotic planetary missions.

Few people are as qualified to get a robot on the moon as Red Whittaker. The 63-year-old may have done more than any other individual in developing the discipline of field robotics—taking robots out of controlled environments such as automobile factories and releasing them to do useful work in the wild. In the 1980s he designed and built the robots that explored damaged and dangerously radioactive areas of the partially melted-down Three Mile Island nuclear power plant. As founder and head of the Field Robotics Center at Carnegie Mellon, Whittaker has since made a career of breaking new ground in autonomous vehicles. He has created robots that hunt meteorites in the ice fields of Antarctica and robots that climb into the craters of active volcanoes in Alaska and Antarctica.

Whittaker began planning for the Google Lunar X PRIZE in 2007 while in the midst of a different competition: the Defense Advanced Research Projects Agency's Urban Challenge, held at the former George Air Force Base in Victorville, Calif. Under the

team name "Tartan Racing," Whittaker and his students partnered with General Motors, Continental and other sponsors to create a driverless Chevy Tahoe named "Boss." Even as he won a first-place victory in the world's first autonomous vehicle race through city streets, Whittaker wasted no time in finalizing plans for a class at Carnegie Mellon called Advanced Mobile Robot Development. The class's modest objectives, as described in the course catalogue, are to "detail, analyze and simulate a robotic lunar lander, field-test a lunar rover prototype, tackle enterprise challenges, and communicate mission progress through writing, photography and video." The course is open to Carnegie Mellon students of any field at any level. Around the same time, Whittaker established Astrobotic Technology as a for-profit company with long-time space entrepreneur David Gump at the helm. Gump aggressively pursues corporate sponsorships and potential customers, whereas Whittaker contributes deep knowledge accumulated over more than 29 years of research at the Field Robotics Center. Among Astrobotic's sponsors is Pittsburgh-based Alcoa, which has donated the aluminum required for the spacecraft that will carry the rover to the moon.

Whittaker, an ex-marine and the son of a chemist and an explosives salesman, says that landing one of his team's creations on the moon would represent the fulfillment of a career path that has seen his robots on land, water, underwater, underground, and in just about every environmental extreme here on Earth. Winning the moon doesn't just mean the first prize; in his mind, Astrobotic won't be successful until it meets every one of the bonus objectives as well. "If you haven't done everything," he says, "you haven't done anything."

ROCKET SCIENCE

WHITTAKER'S VISION for getting Astrobotic's spacecraft and rover on the moon begins with the SpaceX Falcon 9 rocket. Established with the goal of dramatically reducing the cost of space access, SpaceX may be the key enabler of the Google Lunar X PRIZE competition. Whittaker believes that the SpaceX rocket will be the vehicle of choice for all the teams in the competition. "As far as I'm aware, every U.S. contender is targeting SpaceX," he says. Even so, the cost of launch will be the single greatest expense for any team. Though less expensive than other rockets in its class, the published price of a Falcon 9 launch is still \$54 million—more than twice the top prize. SpaceX's competitors are reluctant to discuss their own launch arrangements, but it is clear that SpaceX has already upended the market with the single biggest commercial launch contract in history—a \$492-million deal with Iridium, a satellite communications company.

After Red Rover leaves Earth's atmosphere atop its Falcon 9, the Astrobotic spacecraft-and-rover stack will jettison its protective nose fairing, and the rocket's second-stage engine will push the spacecraft and rover on a course to the moon. The transit will take five days. Guidance, navigation and control software developed at Carnegie Mellon will keep the rocket on the right path. The software is a direct descendant of the code that enabled Tartan Racing to win the Urban Challenge. The computational challenges of autonomous driving and spacecraft piloting are not so different—the same kind of math solves both problems, which is why the software is so similar. The main difference, says Astrobotic team member and Ph.D. candidate Kevin Peterson, is the lack of GPS to guide the vehicle. Instead the craft will plot its trajectory to the moon by referencing stars, the moon and Earth.

Once in orbit, the spacecraft and rover must descend to the moon's surface. In 1969 astronaut Neil Armstrong piloted the lunar module from orbit to a specific location on the moon,

while avoiding local hazards such as boulders and craters. But the 250,000-mile distance between our planet and its satellite imposes a time lag that precludes real-time control by a pilot on Earth, so the spacecraft's software will have to accomplish autonomously what Armstrong did by hand. A primary descent engine will burn to slow the spacecraft down as it approaches the moon, while small thrusters will keep the vehicle stabilized. Touching down two days after lunar dawn, the lander will deploy two ramps (the second is a spare, in case a rock or crater obstructs the first). The bolts that hold the ramps folded against the ladder are rigged to break apart under intense heat. After the ramps fall from the spacecraft to the ground, the rover will roll down one of them to the moon's surface, binocular eyes scanning the ground ahead. Moon dust is too slippery to permit an accurate reading of distance traveled based on how many times the rover's wheels have turned. Instead the rover's on-board computer will calculate distance by comparing the changing appearance of surface features as the robot moves. Radiation-hardened components will protect the computer from the unfiltered solar and cosmic radiation with which the airless moon is bombarded.

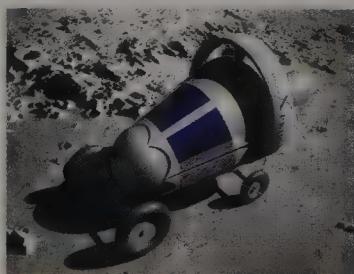
Back in Pittsburgh, Astrobotic team members at mission control will work 24-hour shifts through the long lunar day, using a steady stream of low-resolution images to guide Red Rover to interesting features (including, it is hoped, an *Apollo* landing site). The rover will avoid hazards on the moon's surface autonomously. It will beam high-definition video as blocks of encrypted data, at least one immediately after landing and one later in the mission to meet X PRIZE requirements. The rover will also send e-mail, tweets and Facebook posts.

A major technical challenge for the team is making sure Red Rover survives the extremes of lunar day and night, each of which lasts two Earth weeks. During the two-week lunar night, the temperature at the moon's surface where the team plans to land plummets from a daytime high above 248 degrees Fahrenheit to around -274 degrees F. Any components that contained traces of water, such as the batteries, would suffer irreparable damage as the water froze and expanded. The only rovers ever to have survived the extremes of day and night were the Soviet remote-controlled lunar rovers, called Lunokhods, in the 1970s. They relied on a radioactive polonium isotope to stay warm. But Astrobotic and other private companies competing for the X PRIZE do not have access to these tightly controlled materials. To protect Red Rover from the heat of the sun, carbon-fiber

SPACECRAFT

Rover Reconnaissance

Twenty-six nongovernment teams from around the world are competing in the Google Lunar X PRIZE. Each team must design and build a rover, get that rover to the moon and guide it around the lunar surface—all by the end of 2015. The visions of some teams follow.



Euroluna

Euroluna plans to build a four-wheeled, solar-powered rover that weighs a mere 110 pounds.



JURBAN

JURBAN's small, autonomous swarm bots link up like a centipede or split up to complete different tasks.



Odyssey Moon

Odyssey Moon was the first team to register for the X PRIZE. Its lunar lander builds on NASA technology.

structures surrounding the battery cells conduct heat to the outer surface of the rover. At night, Red Rover will hibernate, and it will awaken with the sun to fire up nonaqueous lithium iron phosphate batteries rigorously tested by then Carnegie Mellon mechanical engineering undergraduate Charles Muñoz.

That is the kind of innovation on the cheap that the X PRIZE is meant to inspire. Although Astrobotic stands a good chance of winning the Google Lunar X PRIZE race, it faces steep competition from India and Russia, which are jointly sponsoring a lunar rover, and from China, which is building a rover of its own that will use a radioisotope to stay powered up through the lunar night. If one of these gets to the moon first, the top prize drops to \$15 million.

COMPETITION

WHITTAKER'S TEAM is also expecting strong competition from other X PRIZE participants. Mountain View, Calif.-based Moon Express, with backing from billionaire co-founder Naveen Jain and other wealthy individual investors, may be the best funded of the Google Lunar X PRIZE teams. It entered the fray only in 2010, three years after the contest was announced, so it is lagging behind Astrobotic. But it is overcoming its latecomer disadvantage with a preexisting spacecraft platform developed by NASA. Another contestant is Boulder, Colo.-based Next Giant Leap, headed by former U.S. Air Force pilot-turned-entrepreneur Michael Joyce. Joyce's company has teamed up with Draper Laboratory (which designed the guidance, navigation and control systems that shepherded the *Apollo* spacecraft to the moon), a group at the Massachusetts Institute of Technology, and the space systems branch of Sierra Nevada Corporation. It is building a novel "hopping" spacecraft that obviates the need for a separate rover. The craft reignites the thrusters it uses for touchdown to lift off again and travel short distances to areas of interest. The idea seems workable but only if Joyce can raise the necessary funds.

The Google Lunar X PRIZE organizers hope that if they build it, the market will come—that developing rovers and getting them on the moon will spur the growth of a new market. Astrobotic, for example, is offering room onboard its spacecraft and rover at the rate of \$1.8 million and \$2 million per kilogram (2.2 pounds), respectively, plus a \$250,000 "integration fee." For researchers such as University of Maryland physicist Douglas Currie, at least, a guaranteed spot for a fixed price on a commercial mission would be a boon. Currie and his colleagues

The most enduring benefit of the Google Lunar X PRIZE may be inspiring the next generation of scientists and engineers.

want to place an array of laser-ranging retroreflectors on the moon to support measurements that would be 100 times more accurate than can be made with those left by the *Apollo* astronauts—if only missions become available on which to fly them.

Perhaps the most enduring benefit of the X PRIZE will be to inspire the next generation of scientists and engineers. The race has lent an air of real-world excitement to Whittaker's Advanced Mobile Robot Development course. During the final week of classes in April 2011,

members of the Astrobotic structures team scurry about the 3,000-square-foot workshop of Carnegie Mellon's Planetary Robotics Laboratory, which is entirely dedicated to the moon rover project. They are testing the design for fragmenting metal bolts, an alternative to typical explosive bolts, that unhinge the ramps from the spacecraft so that the rover can explore the lunar surface. Grad student Kanchi Nayaka and a group of undergrads prepare a high-speed video camera on a tripod to record the simulation. The students then throw a switch, and 17.9 seconds later the bolt breaks apart with a bang, and the ramp swings open and falls to the ground, ready for the rover to emerge.

"Awesome!" Nayaka says. She steps back from the camera and shoots a grin at a visitor. "You must be good luck!" ■

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SCIENTIFIC AMERICAN ONLINE

To view a gallery of images related to Astrobotic Technology and the Google Lunar X PRIZE, visit ScientificAmerican.com/apr2012/x-prize



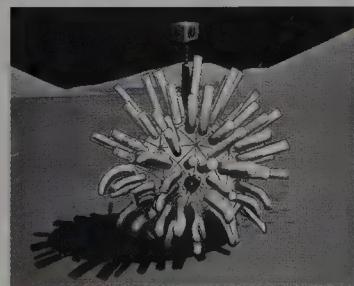
Synergy Moon

Spectators on Earth can control Synergy Moon's spherical rover as it surveys the moon with twin cameras.



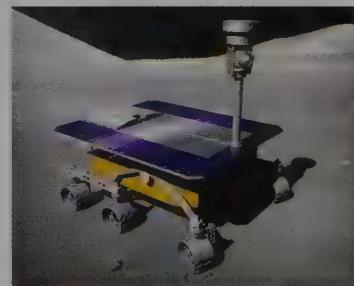
Italia

Italia has not yet finalized the design for its rover, but the spider bot pictured here is one option.



Puli Space

Puli Space's urchinlike rover evokes the dreadlocks of the Hungarian dog breed after which the team is named.



White Label Space

The wheels on White Label Space's rover are designed to not slip on the moon's dusty surface.

PUBLIC HEALTH

POLIO'S LAST ACT

As the number of cases of the paralytic disease fall, world health officials have to grapple with a vexing problem: a component of the most widely used polio vaccine now causes more disease than the virus it is supposed to fight

By Helen Branswell

The global campaign to eradicate polio began in 1988. Since then, naturally occurring cases worldwide have dropped to, at last count, around 650 in 2011.

Completely eliminating polio requires a change in the current vaccination program because one component in the most widely used vaccine now causes more cases of polio than it prevents.

The World Health Assembly is expected to approve a plan this May that should decrease the number of vaccine-linked cases of polio and may speed up overall eradication efforts.

Yet questions have arisen over the safety of making the change rapidly. If health officials do not manage this transition correctly, polio could continue to cripple children for years to come.



WHO IS AT RISK?

Although polio has disappeared from the Western Hemisphere and Europe, the virus still permanently cripples children in Africa and Asia every year.

Helen Branswell is the medical reporter for The Canadian Press, where she has covered developments in the effort to eradicate polio since 2004. She was a Nieman Global Health Reporting Fellow in 2011. The travel and research for this article were supported by the Pulitzer Center on Crisis Reporting and the Nieman Foundation for Journalism at Harvard University.



T

HE SHADOWS LENGTHEN IN A GUESTHOUSE CAFETERIA ON THE SPRAWLING CAMPUS OF CHRISTIAN Medical College, Vellore, in India. Wrapped up as he is in an issue that has possessed him for years, T. Jacob John notices neither the dying light nor the gathering mosquitoes. He is talking about the oral polio vaccine.

A slight man who speaks and moves with a speed that belies his 76 years, John is one of India's leading polio experts. Trained as a pediatrician, virologist and microbiologist, he is also a long-time critic of the continued reliance on the oral polio vaccine—OPV in polio speak—used by the nearly 25-year-old international campaign to rid the planet of the paralyzing and sometimes fatal disease. The vaccine is at once an excellent and an imperfect tool. Inexpensive and easy to administer (each dose consists of a few drops of serum on the tongue), it has brought the world to the point where polio eradication is visible on the horizon. Indeed, the World Health Organization announced this past January that there have been no cases of naturally occurring polio in India for a year. But if the distribution of the vaccine is not choreographed with exquisite care, its continued use—at least as it is currently formulated—could actually keep the world from eliminating polio.

DOWNTURN TRENDS

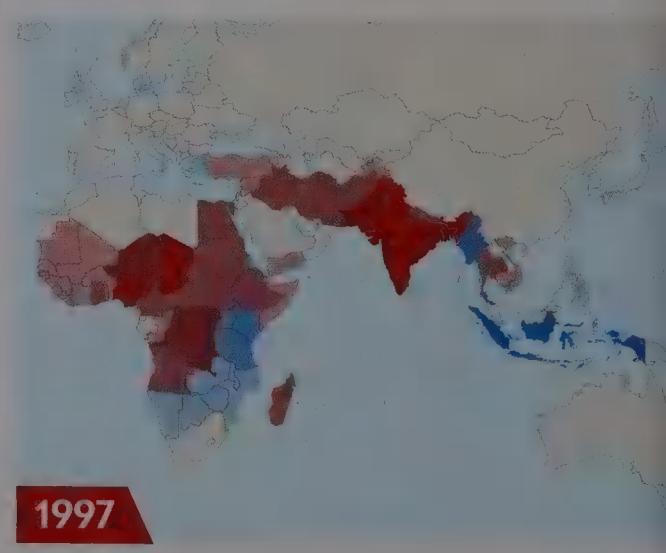
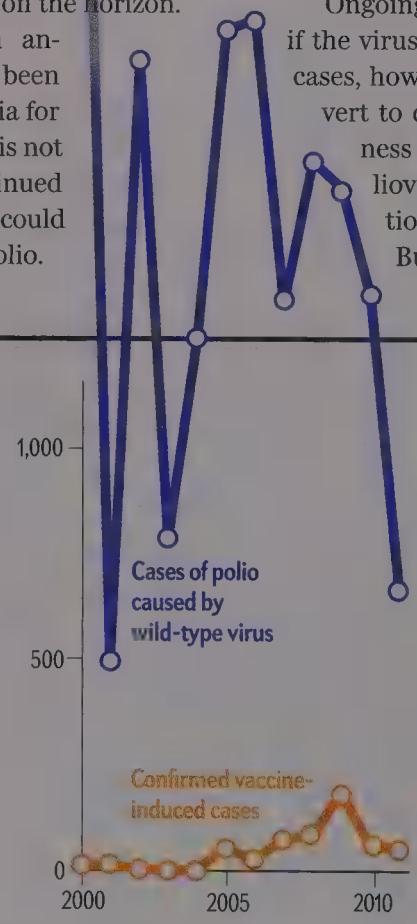
Polio Progress

Polio once crippled hundreds of thousands of youngsters. Widespread use of the oral polio vaccine has brought the number of cases down dramatically, and by early 2012 eradication efforts had limited the number of countries where polio outbreaks are endemic, or self-sustaining, to three. As the number of naturally occurring infections drops, however, the rare instances of vaccine-related polio cases become less tolerable, necessitating a change in vaccine strategy.

Today John is talking with a reporter about a problem raised by a specific component of the oral vaccine, which uses weakened viruses to elicit immunity against the three strains of polio—known as types 1, 2 and 3. (An expensive, alternative vaccine, popular in wealthy nations, consists of an injected formulation that is made up of completely inactivated, or “killed,” viruses; it is known as IPV.) The issue: type 2 poliovirus no longer exists in nature; the last case stemming from naturally circulating virus was reported 13 years ago.

Ongoing vaccination against type 2 would not be worrisome if the viruses in the oral vaccine were perfectly benign. In rare cases, however, the weakened viruses from the vaccine can revert to disease-causing pathogens and provoke the very illness they are meant to prevent. In places where wild polioviruses are still a threat, the risk from natural infection is greater than the small hazard the vaccine poses. But if the only risk of paralysis from type 2 polio comes

SOURCES: WORLD HEALTH ORGANIZATION (graph); GLOBAL POLIO ERADICATION INITIATIVE AND WORLD HEALTH ORGANIZATION (maps)



from the strain in the vaccine itself, then that strain's continued usage could well be considered unproductive at best and quite possibly unethical. As long as the oral vaccine contains the type 2 virus, however, children in more than 100 countries around the globe must—paradoxically—be vaccinated against type 2 polio to protect them from the type 2 virus in the vaccine.

In 2004 John wrote a letter to the medical journal the *Lancet*, urging the international community to remove the type 2 component from the oral vaccine, thus making it a “bivalent” vaccine that would protect against types 1 and 3 polioviruses. Like other complaints John has made about the oral polio vaccine, however, the suggestion went nowhere—until now.

The Global Polio Eradication Initiative—a partnership of the WHO, UNICEF, Rotary International and the Centers for Disease Control and Prevention—is marshalling support for an initiative to drop the type 2 component from the oral vaccine. The proposal is part of a substantial overhaul of the plan to eventually phase out the oral polio vaccine altogether once all types of wild polioviruses are demonstrated to have been extinguished. The WHO's governing council, the World Health Assembly, will be asked to approve the early withdrawal of the oral vaccine's type 2 component at its annual meeting in May.

If the policy change passes—and the assembly is expected to vote in its favor—the move would eliminate an ethical problem that has been bedeviling the eradication effort for years. It could also speed the job of wiping out the remaining two strains of polio in the three countries where they remain endemic (Afghanistan, Pakistan and Nigeria); a 2010 *Lancet* study showed that the two-target vaccine is at least 30 percent more effective than the one that has to protect against three strains of polio. And yet the poliovirus has a nasty habit of eluding efforts to contain it. Last year, for example, China reported its first cases—genetic tests traced their origin to Pakistan—in more than a decade. Adjusting the oral polio vaccine, some fear, could have unintended consequences and thus disrupt an eradication campaign that is already 12 years past its original deadline and counting.

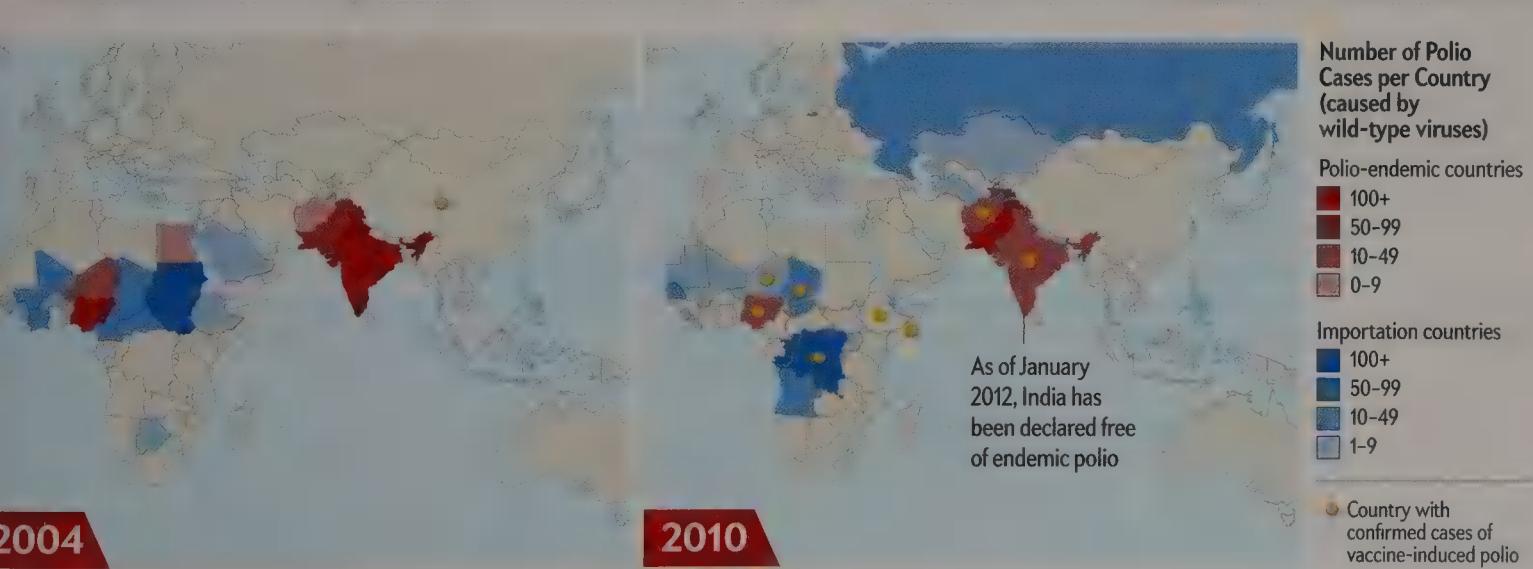
CHANGING TACTICS

COUNTRIES HAVE USED the injected and oral vaccines to protect their citizens against polio for half a century. Jonas E. Salk de-

veloped the killed-virus vaccine, which was licensed by the U.S. in 1955, and Albert B. Sabin developed the oral vaccine, which was fully approved in the U.S. in 1962 [for newly available details about Sabin's efforts, see “Birth of a Cold War Vaccine,” by William Swanson, on page 66]. The greatest advantage of the oral vaccine—besides its low cost (about 15 cents a dose compared with \$3 a dose for the injectable vaccine)—is its ability to trigger a low-level and generally safe infection that prompts the immune system to respond, thereby immunizing the recipient. An added bonus is that vaccinated children excrete vaccine viruses in their stool; in households, playgrounds and communities, those vaccine viruses spread from vaccinated to unvaccinated youngsters, eliciting a protective immune response in children who have not been inoculated. Health authorities had known from the early 1960s that Sabin's live-virus vaccine would occasionally paralyze a child who received the drops (or even more rarely their immediate contacts), but they felt the millions protected justified that unfortunate cost. (The idea that vaccine viruses can also circulate on their own, causing problems in large groups of unvaccinated children, was not recognized until much later.) Eventually most countries adopted Sabin's oral polio vaccine to protect their children—although some, such as the U.S., later switched back to Salk's injected formulation.

For years the global eradication strategy was pretty straightforward: use the oral vaccine in the countries that preferred it or could not afford the more expensive inactivated polio vaccine until wild polioviruses were declared gone. Then, at a pre-arranged time, all countries would simultaneously stop using the oral vaccine. Wealthy countries would undoubtedly continue to vaccinate with the killed-virus vaccine for a time as further protection against an unexpected reemergence, but if developing countries could not afford that option, then their children would go without vaccination—and the world would hold its breath waiting to see if polio was truly gone.

Over the past decade many polio experts have argued against that plan, calling it a high-stakes experiment that would put millions of children at risk. Now, it seems, the so-called cold turkey approach may finally be coming off the table. “Most people have moved away from cold turkey,” says Roland W. Sutter, the



WHO scientist who heads research policy and product development efforts for the Global Polio Eradication Initiative. Promising research on the effectiveness of ultrasmall doses of inactivated polio vaccine is creating the hope that eventually a tiny, or "fractional," dose of injectable polio vaccine could be bundled into a six-in-one childhood vaccine that would offer the world's youngsters protection against diphtheria, tetanus, pertussis, hepatitis B, *Hemophilus influenzae* B and polio, Sutter says. But that objective would be considered sometime down the road.

For now the focus is on safely eliminating the type 2 virus from the oral polio vaccine. In addition to the ethical issues raised by retaining the type 2 component, the WHO and other health agencies are grappling with another concern: the component is standing in the way of completely eliminating polio. "The real driver of this is, How do you accelerate eradication?" says R. Bruce Aylward, the indefatigable Canadian who has long led the WHO's polio effort. The answer to that question, he and his team have concluded, is to find a way to phase out the three-part ("trivalent") oral vaccine using a formulation targeting types 1 and 3 instead. They expect to see immediate benefits, given that the two-strain version is more effective than the trivalent vaccine. Indeed, that is why India and Nigeria have been using a two-strain vaccine in some immunization rounds for the past couple of years. (Children in high-risk areas are often vaccinated many times to build their immunity.)

A TRICKY TRANSITION

GLOBAL OFFICIALS are increasingly feeling the need to alter the trivalent vaccine because the cases of paralysis attributed to the type 2 vaccine component become harder to justify as the number of naturally occurring cases continues to decrease. Years of experience with the oral vaccine have shown that two to four out of every one million children born in the same year will develop polio from the oral vaccine, with roughly 40 percent of these cases caused by the type 2 viral component. (A child's risk of contracting polio from the vaccine falls with each additional dose he or she gets.) All in all, the WHO estimates that about 120 children

get polio every year as a result of the inoculation, although John thinks the true number is several times higher than that figure.

In addition, there are the rare indirect deleterious effects of polio vaccination. From 2000 to 2010 the secondary spread of vaccine viruses from vaccinated to unvaccinated children led to at least 538 cases of polio. The type 2 vaccine virus was responsible for 84 percent of those secondary cases.

When the vaccine itself causes polio, the resulting malady is called vaccine-associated paralytic poliomyelitis, or VAPP; when a nonvaccinated person contracts polio from vaccine viruses that are spreading from person to person, the virus is termed vaccine-derived poliovirus, or VDPV.

The biggest outbreak of vaccine-derived polio began in 2005 and is still under way in Nigeria, where the spread of the type 2 vaccine virus among unvaccinated children has crippled at least 376. Vaccine-derived viruses from that epidemic have also spread to nearby Niger and Guinea. The Nigerian outbreak will have to be halted before the world can safely drop the type 2 component from the oral polio vaccine, Aylward says. In a sad twist of fate, vaccination with a formulation that includes the type 2 strain has to continue despite its risks because for now it is the most feasible way to confer immunity to that strain.

Once the type 2 component can be safely removed, health officials foresee a transition period when first the injected and then the two-strain oral vaccines are used in successive waves. The killed-virus vaccine is needed to drive up immunity levels to the type 2 virus in case any residual viruses of this strain are still circulating. Health authorities hope to keep the price down below what a full-fledged injected vaccine campaign would cost by giving all children one or two fractional doses of the inactivated formula. Research suggests that under certain circumstances—and when done sequentially with the application of the oral vaccine in the same person—splitting doses of inactivated vaccine should be as effective as providing full-potency injections.

The cost of the injectable vaccine needed to prepare for the across-the-board move to the bivalent vaccine could be brought down to 35 or 40 cents a dose with bulk buying, local manufac-

VACCINE STRATEGIES

Tools for the Endgame

Public health officials have used two major types of vaccines to protect children against polio—one made from live but weakened virus and the other made from killed virus. The risks and benefits of the two kinds of live-virus vaccine, as well the killed-virus vac-

cine, are detailed below. Eliminating the disease will require making a tricky transition from the widely used live vaccine with three components to new versions with two components and ultimately to withdrawing the live-virus vaccine altogether.

	Description	Pros	Cons
Trivalent live vaccine (tOPV)	Contains weakened versions of all three strains of poliovirus (types 1, 2 and 3)	<ul style="list-style-type: none"> Delivered by mouth, so only minimal training is needed to administer the vaccine Inexpensive Nonvaccinated children can benefit 	<ul style="list-style-type: none"> In rare cases, weakened virus from the vaccine can cause paralytic polio; natural type 2 virus no longer circulates, so now all cases of type 2 polio come from the vaccine itself
Bivalent live vaccine (bOPV)	Contains only types 1 and 3 polioviruses	<ul style="list-style-type: none"> All the benefits of trivalent vaccine but will not cause type 2 polio 	<ul style="list-style-type: none"> Unsafe to use if type 2 vaccine poliovirus is still circulating undetected
Killed-virus vaccine (IPV)	Contains chemically inactivated versions of all three poliovirus strains	<ul style="list-style-type: none"> Does not cause polio 	<ul style="list-style-type: none"> Expensive Injected, so must be administered by health care personnel (who are scarce in poor countries)

ture (in places such as India and China) and fractional dosing, the WHO's Aylward says. Economic analyses suggest that if the price per injection can be reduced to 50 cents a dose, using the inactivated vaccine becomes feasible even in very poor countries. The bottom line is that a supercheap version of the injected vaccine—at least supercheap relative to the usual formulation—"all of a sudden changes the ball game," Aylward says. "You're taking cost away from the discussion, and you're having a scientific discussion and a programmatic discussion about what is the safest way to manage the risk of polio these days."

BUILDING ON EXPERIENCE

TRANSITIONING BETWEEN polio vaccines is a complex undertaking even in countries, such as the U.S., with substantial resources. Many officials were staunchly opposed when the U.S. first started considering a switch back to Salk's inactivated vaccine in the early to mid-1990s. They feared the move would backfire badly.

Relying strictly on the inactivated vaccine would mean that some nonvaccinated individuals who previously would have gained protection from the spread of the vaccine virus would no longer be afforded that passive protection. They would be vulnerable to infection from either travelers who harbored the poliovirus or from vaccine viruses from people who had been immunized with the live-type vaccine. "It took a good bit of time to convince people that in a nation like this, where we were so successful in our immunization program, that we could switch to IPV," says Samuel L. Katz, an emeritus professor of pediatrics at the Duke University School of Medicine, a co-developer of the measles vaccine and, until about a year ago, chair of the WHO's Polio Research Committee.

Walter Orenstein, a polio specialist now at Emory University who has worked with the CDC and the Bill & Melinda Gates Foundation, was an ardent supporter of the oral polio vaccine. He was convinced the U.S. had only managed to rid itself of the scourge of polio because of the secondary spread of Sabin's vaccine. "I was scared that if we switched to IPV only that we would still have this risk of new polio outbreaks," Orenstein explains. He knew the vaccine paralyzed a few children every year but worried that if a switch to inactivated polio vaccine left an immunity gap through which polioviruses could slip, many times more children would be hurt.

At one of many meetings held to debate whether the U.S. should switch back to the inactivated polio vaccine, Orenstein had a religious-style conversion. The year was 1996; it had been 17 years since polio had spread within U.S. borders. People who had been crippled by polio vaccine viruses attended the meeting to press for the change. "When I looked at those people and recognized that had they gotten IPV, they would never have gotten paralyzed and would have likely been protected from polio, it was hard to continue," Orenstein admits. "It was a defining moment. I became an IPV advocate." The U.S. began a gradual phasing out of the oral vaccine in 1997 (it inoculated children with both vaccines in tandem through 1999 to guarantee that an adequate level of public protection was maintained).

Orenstein vividly recalls the uncertainty of the time. "Had we been wrong, had these fears been played out and we had a big polio outbreak in the U.S., people would have said, 'Why in the world did you switch?' It's a lot easier to be a historian than it is to be making policy. Because there were a lot of unknowns."

THE ENDGAME

EXPERIENCE SOON PROVED the U.S. had made the right decision. Within months of switching to inactivated polio vaccine, the number of vaccine-associated cases of polio dropped to zero. Currently 56 countries use the inactivated vaccine exclusively. (Some countries, such as Sweden, always opted for the safer but more expensive, injectable vaccine.) Sixteen countries immunize with both vaccines, and 121 countries use the oral vaccine alone.

The switch from trivalent to bivalent oral vaccine could come sometime between April 2013 and April 2014, says Sutter of the Global Polio Eradication Initiative. A number of things have to fall into place before the move can be made, including ensuring that countries that make their own oral polio vaccine—such as Mexico, China and Brazil—quickly move to license a two-strain oral vaccine. If they require manufacturers to conduct additional safety and efficacy trials of the newer vaccine rather than relying on a WHO-led clinical trial that has already been completed, the changeover could be delayed.

Major questions remain. Would fractional doses of inactivated vaccine be introduced everywhere, just in high-risk countries, or perhaps in high-risk countries and their neighbors? Unlike the oral vaccine, the injected formulation must be given by a health care professional, and many of the countries on the polio front line also have shortages of trained personnel—hence, the need to marshal resources wisely. Orenstein, who is involved in the discussions, says the details are still being worked out. For now the WHO's member countries are only being asked to approve the withdrawal of oral vaccine virus components in phases beginning with what WHO documents describe as "the particularly problematic Sabin type 2 poliovirus."

Will development of a two-strain oral vaccine satisfy John, who pushed for this change eight years ago? Ironically, he is now one of the people concerned about the plan. Having seen how readily vaccine-derived polioviruses spread from person to person, he now believes that leaving children without protection to the type 2 virus in the next few years would be unsafe because some of the vaccine-derived virus may yet linger undetected in the environment. John says he would feel comfortable making the switch only after there is solid evidence that the preparatory campaign with inactivated vaccines has resulted in very high, widespread levels of immunity against type 2 poliovirus. Otherwise, he fears, even the painfully slow progress of the past few years will vanish, and polio will continue crippling children for years to come. ■

MORE TO EXPLORE

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Centers for Disease Control and Prevention page on Polio Disease—Questions and Answers: www.cdc.gov/vaccines/vpd-vac/polio/dis-faqs.htm

Global Polio Eradication Initiative: www.polioeradication.org

SCIENTIFIC AMERICAN ONLINE

See photographs from Helen Branswell's reporting trip to India and listen to her talk about her experience at ScientificAmerican.com/apr2012/branswell-india

HISTORY OF SCIENCE

BIRTH OF A COLDWAR VACCINE

While the superpowers were busy threatening to destroy each other with nuclear weapons, Albert B. Sabin turned to a surprising ally to test his new oral polio vaccine—a Soviet scientist

By William Swanson

TO MANY AMERICANS, THE COLD WAR IS ANCIENT HISTORY. Yet only a few decades ago the planet was dangerously divided between West and East, and the antagonism between the U.S. and the Soviet Union defined global politics. Flare-ups such as the Korean “police action,” which killed millions of people in the early 1950s, and the Cuban missile crisis, 10 years later, drew the American and Soviet governments and their proxies to the threshold of nuclear war.

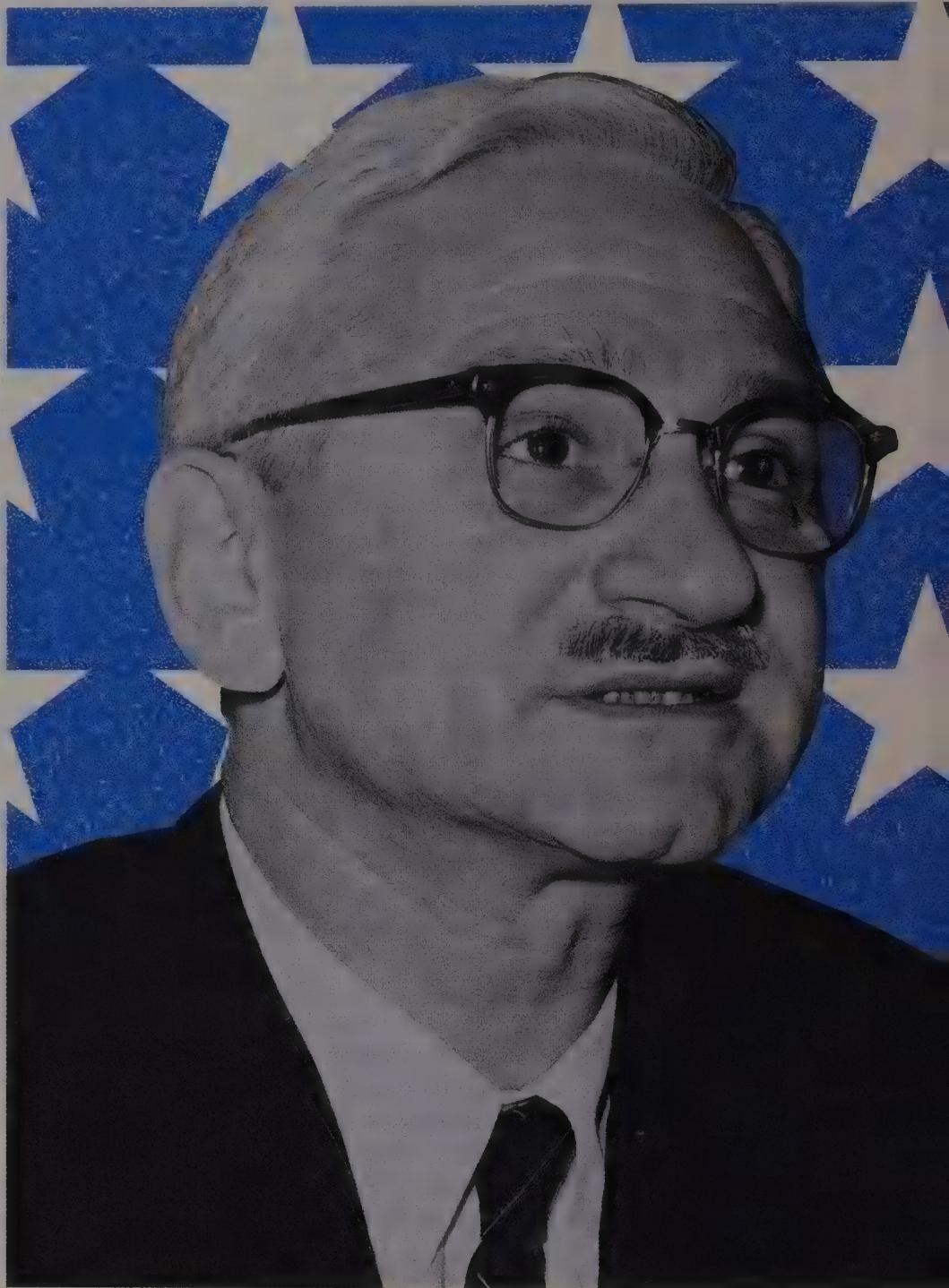
At the same time, Americans lived in mortal fear of an enemy much closer to home. That enemy was polio—short for poliomyelitis, also known as infantile paralysis because of its prevalence among children and young adults. Scientists had known its cause—a virus spread via contact with fecal matter—since the 1930s, but its control eluded them. During sporadic epidemics authorities closed swimming pools, movie houses and other popular gathering spots, hoping to contain the disease, which attacked the central nervous system, often crippling and some-

times killing its victims. Newsreel footage of toddlers with twisted limbs and teenagers lying helplessly on their backs in coffinlike iron lungs frightened the public as few of the era’s images did.

Then, during the deep winter of the cold war, two extraordinary scientists—one an American, the other a Russian—formed a powerful alliance. Their joint venture would have outraged fanatics on both sides of the iron curtain if those fanatics had been aware of it. Yet the collaboration—fleshed out in archival materials recently made available at the University of Cincinnati and by several contemporaneous sources—led to one of the greatest medical achievements of the 20th century and saved countless lives around the world.

THE QUEST FOR AN EFFECTIVE VACCINE

BY THE EARLY 1950S the quest for a polio vaccine had moved into high gear in the U.S. Virologists Jonas E. Salk of the University of Pittsburgh and Albert B. Sabin of the University of Cincinnati





ALLIES: Although their countries were at odds, Albert B. Sabin (*opposite page*) and Mikhail P. Chumakov showed that an oral vaccine could protect millions against polio.

had emerged as the most prominent among dozens of American researchers funded by the National Foundation for Infantile Paralysis (now the March of Dimes Foundation). In 1955, after tests involving nearly two million schoolchildren across the country, Salk's vaccine became the first to receive the approval of the U.S. government. While Salk became, in the words of historian David M. Oshinsky, "an instant hero, a celebrity-scientist," Sabin continued to labor over what he believed was a superior vaccine.

Both approaches would protect against infection, though in different ways. The Salk vaccine was made up of polioviruses that had been inactivated, or "killed" in popular terminology, using the chemical formalin. Sabin thought that a vaccine made up of a weakened but still active poliovirus would be more effective than a killed-virus vaccine because it could generate lifelong immunity.

strains of poliovirus he believed had to be included to make his vaccine effective (Salk had used different strains), but he lacked the numbers to prove himself correct. Beginning in the mid-1950s, Sabin tested his vaccine on hundreds of volunteers, including young adults incarcerated at the federal prison in Chillicothe, Ohio, as well as himself, his wife and their two daughters, neighbors and friends. (He assured the prison volunteers that they faced considerably less risk ingesting his vaccine than he had faced driving from Cincinnati in a snowstorm.) Even as he conducted the tests, though, he knew that hundreds, even thousands, of study participants would not be enough. He needed millions of subjects to document his vaccine's safety and efficacy. Because the Salk vaccine was already widely used in the U.S., there were not enough unvaccinated Americans to provide sufficient numbers.

William Swanson is a Minneapolis-based freelance journalist whose topics include health, history and politics.



Live-virus vaccines also offered the possibility of secondary immunization, in which vaccinated children would passively infect their contacts with the vaccine virus, thereby immunizing many unvaccinated people. Finally, unlike Salk's vaccine, which was injected, the Sabin vaccine could be administered in a bite-size sugar cube or swallowed off a spoon. Thus, tens of thousands, even millions, of citizens could be given the vaccine quickly, inexpensively, and without the fear and fuss of needles. For all these reasons, Sabin believed that the best hope of not only controlling the disease but wiping it off the face of the earth lay with his live-virus oral vaccine.

Mainstream media made much of the competition between Sabin and Salk even though other important scientists were also involved in the "race" to capture the U.S. market. There was a certain amount of truth underlying the press's exaggerations. Salk and Sabin—despite their common Russian-Jewish heritage, funding source and virological enemy—disliked each other intensely. Sabin scoffed at Salk's "kitchen chemistry," insisting that Salk "didn't discover anything." Salk believed that Sabin, jealous of his early success, was "out for me from the very beginning." Sabin doubtless resented the enormous acclaim Salk enjoyed following the dead-virus vaccine's approval, whereas Salk surely bristled at the suggestion that, in the words of one contemporary, he "was an overblown publicist, while Sabin was the real scientist."

By 1955 Sabin had identified the three

Meanwhile, in the Soviet Union, the incidence of polio was rising sharply. For years during the dictatorship of Joseph Stalin, authorities denied that polio was a problem in the "workers' paradise." But as outbreaks in Moscow, Minsk and population centers as far away as Siberia put the lie to the propaganda, Soviet scientists sought the same answers pursued by their U.S. counterparts. Soviet and American investigators had occasionally joined forces in the quarter of a century between the 1917 Bolshevik revolution and the end of World War II. That cooperation all but vanished, however, when East and West squared off in the aftermath of the Allied victory in 1945. Then, in 1953, Stalin died, and his slightly less rigid successors, alarmed by the increasing polio numbers, directed their researchers to look beyond their borders for help.

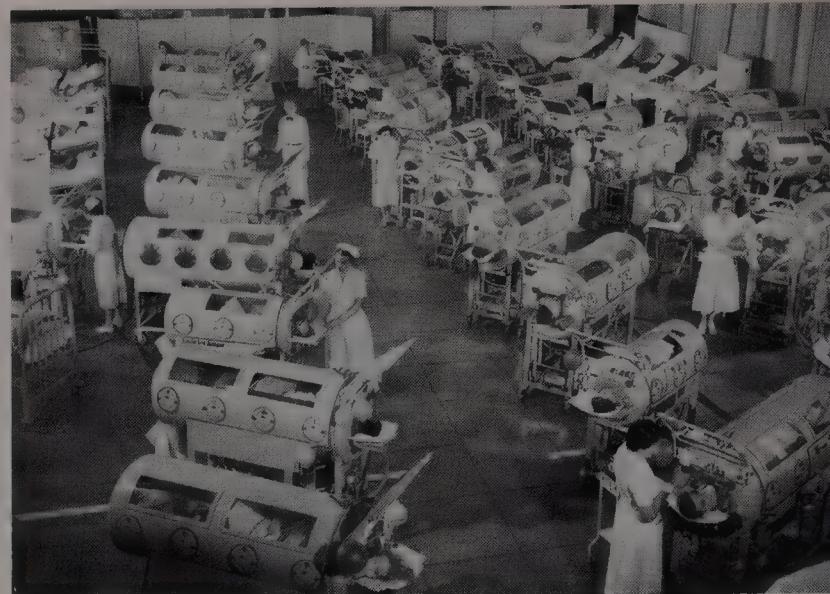
Russia's two most prominent virologists at the time were Anatoli Smorodintsev and Mikhail P. Chumakov. In January 1956 Smorodintsev, Chumakov and Chumakov's wife, Marina Voroshilova—a distinguished researcher in her own right—traveled to the U.S. to confer with several American scientists, including Salk and Sabin. Though quietly approved by both governments, the visit was shadowed by cold war bugbears: the Russians were required, for instance, to cross the country by rail rather than, more conveniently, by air, and the Americans were convinced that at least one "doctor" accompanying the visitors was a KGB operative. Still, both sides discreetly hailed the tour as a success. Valuable scientific information was exchanged; more important, as events transpired, Chumakov and Sabin hit it off, establishing the ties that would lead to a spectacularly productive relationship.

DR. SABIN GOES TO RUSSIA

IN JUNE 1956, authorized by a cautiously obliging U.S. Department of State and vetted by the ever watchful FBI, Sabin flew to Russia and, over the next several weeks, met with Chumakov, Voroshilova, Smorodintsev and other key researchers.

Even though both he and Salk had been invited, Sabin was on a mission of one. Decades later Salk's son Peter told Oshinsky that his father had turned down the Russians' invitation because Salk's wife, weary of her husband's frequent absences, had finally "put her foot down." Oshinsky's chronicle suggests another possibility. As a younger man, Salk had been one of thousands of Americans who publicly espoused left-wing causes and thus aroused the FBI's attention. Perhaps Salk feared that a visit to the Soviet Union would be misconstrued. More likely, the "celebrity scientist," whose game-changing vaccine had made him famous and wealthy, believed he had little to gain from a Soviet trip. Unlike Sabin, he had nothing to prove.

Sabin, for his part, was returning to his roots. He was born in 1906 in Bialystok, a Polish city that had been part of imperial Russia and then the Soviet Union. His family was poor. His father, a weaver, was the breadwinner, but his mother, he recalled much later, was "the one with the initiative." After the Sabins emigrated to the U.S. in 1921, Albert quickly mastered the English language



IRON LUNGS: In the 1950s thousands of youngsters were placed in mechanical ventilators such as these because their respiratory muscles were paralyzed. Pressure changes inside the chamber forced the chest to expand and contract, allowing patients to breathe.

and American ways. After earning a medical degree from New York University in 1931, he made a name for himself as a medical researcher in New York City, London and, eventually, Cincinnati, focusing on polio, encephalitis and other neurological diseases. He was among the researchers who challenged the received wisdom regarding the way by which the poliovirus entered the human body. The virus's entryway was not the nasal passages, as Simon Flexner, the "father of polio research," had theorized, but the alimentary canal; after it entered through the mouth, the virus migrated to the digestive tract, where it infiltrated the bloodstream en route to the central nervous system. That knowledge would prove critical to the next step—developing a vaccine that could induce the immune system to attack the virus in the blood.

In the U.S.S.R., Sabin faced a number of new challenges as he huddled with researchers and championed his live-virus vaccine. His exposure to the language as a child notwithstanding, he was never proficient in Russian, nor were most of his Soviet counterparts fluent in English. Interpreters were provided, but the painstaking collaboration surely would have been easier if the scientists had communicated in the same language. It is tempting to wonder, moreover, what fears and biases might have lingered from Sabin's childhood in Bialystok, where Jews lived in constant fear of attack and where, he once remarked, he grew up "thinking of Russian soldiers as murderers." If Sabin harbored such thoughts during his Soviet visits, he apparently kept them to himself. Later, he would insist he was unfazed by the Soviet and American agents who tracked his movements and recorded his public comments.

Despite the complications, Sabin developed valuable work-

IN BRIEF

At the height of the cold war, paralytic polio was one of the few things that frightened Americans and Russians more than each other.

Newly available documents detail the unlikely collaboration between Albert B. Sabin of the U.S. and Mikhail P. Chumakov of the U.S.S.R. to fight the infection.

Together Sabin and Chumakov proved that a vaccine against polio made with weakened strains of the virus was both safe and effective.

A global campaign against polio using the live-virus vaccine has decreased the number of polio cases worldwide from 350,000 in 1988 to around 650 in 2011.

ing—and, in some cases, close personal—relationships with his Russian hosts over the next several years. None proved to be more beneficial than his friendship with Chumakov.

"THE GENERAL" AND THE RED PHONE

CHUMAKOV, IT TURNED OUT, was a perfect match for Sabin. He was born in 1909 to a humble family in the Caucasus. His father was an army veterinarian, his mother a peasant who did not learn to read or write until she was in her 70s. When Chumakov was 16, his son Konstantin says, he went off to Moscow to attend college and was later admitted to both law and medical schools before choosing a career in medicine.

Neither Chumakov nor Sabin suffered fools gladly, and both were convinced that fools were everywhere. Sabin's brilliance as a scientist was rivaled only by his fearsome reputation as a taskmaster and competitor. Fastidious himself, he demanded a fanatical attention to detail from his staff; dead sure of his positions, he publicly challenged his rivals' conclusions. Philip Russell, an eminent virologist and a founder of the Washington, D.C.-based Sabin Vaccine Institute, knew Sabin and many of the investigators who worked in Sabin's labs. Echoing the widespread opinion of both the man and the researcher, Russell says, "Albert was driven and meticulous—a visionary scientist. He was also tough, arrogant and never wrong—even when he was." Sabin's acquaintances might have been surprised to learn that Chumakov may have had the more volcanic personality. In a 1958 letter to Sabin, Chumakov complained about "the intrigues of... cowards and pseudo-specialists," whom he did not hesitate to cite by name.

"Thankfully, they found each other," says Konstantin, who has lived in the U.S. since 1989 and is currently an associate director in the Office of Vaccines Research and Review at the Food and Drug Administration. "Sabin had the vaccine that could save uncounted numbers from death or paralysis, and my father found the way to push it past the bureaucratic obstacles. Sabin called my father 'the General' because he could get things done."

Russian virologists had experimented with Salk's dead-virus vaccine, but Chumakov sought a simpler, less costly, more efficacious way to extend protection against polio across the vast population of the Soviet Union. In 1959 Chumakov decided to organize the first large-scale clinical trials of the oral vaccine made from the live, weakened strains Sabin had developed in the U.S. It would be a monumental undertaking, fraught with problems—beginning with approval from the top.

"Sabin publicly gave credit to my father and the Soviet system whose organization made such large trials possible," Konstantin says. "But I'm not sure my father ever told Sabin the true story behind it. What actually happened—according to my father—went like this:

"My father couldn't get permission for a really big clinical trial. A lot of people in the Health Ministry were opposed to it. He was told, basically, 'We have the Salk vaccine, and it works fine, so there's no reason for you to test the live virus.' Well, my father decided to go around them.

"In the Soviet Union there was a higher authority—the Politburo [then known as the Presidium of the Central Committee], which consisted of a small group of Communist Party officials who could overrule everybody. At the time, Anastas Mikoyan was the Politburo member responsible for public health. Mikoyan was not a medical man—he was a political figure who went back to the revolution.

But he and my father were well acquainted. Mikoyan may have appointed him to head the polio initiative in the first place." Refusing to accept the ministry's decision not to grant permission for the oral vaccine tests, Chumakov picked up one of the red telephones provided for the exclusive use of the most powerful people in the Kremlin—he was *not* among them—and dialed Mikoyan's number.

As Chumakov related the story to his son, he got right to the point and asked Mikoyan's approval to proceed with the live-virus vaccine tests.

"Are you sure this is a good vaccine, Mikhail?" Mikoyan asked. "And that it's safe?"

"Yes," the virologist replied. "I'm absolutely sure."

"Then go ahead," Mikoyan said.

"That was it," says the younger Chumakov, whose account rings true with others familiar with the principals. "The only permission he had was verbal, over that Politburo hotline. Of course, the health minister was unhappy, but there was nothing he could do."

A LASTING SUCCESS

IN 1959 CHUMAKOV TESTED the oral vaccine on 10 million children throughout the U.S.S.R. The Soviets set up vaccination centers not only at hospitals and clinics but in schools, nurseries and other nonmedical locations. Within the next several months virtually everybody under the age of 20—eventually including almost 100 million persons in the Soviet Union and its satellites—received the vaccine, either by medicine dropper or inside a piece of candy, and the outcome seemed to justify the effort. Chumakov was ecstatic about the vaccine's widespread application, and within a year a representative of the World Health Organization (WHO) acknowledged both the vaccine's safety and a significant reduction of paralytic cases.

There were still, to be sure, Western scientists who refused to accept the glowing reports from the other side of the iron curtain. "The general reaction, usually not expressed publicly, was, 'Well, you can't trust anything those people do,'" Sabin grumbled more than once. But the documented achievement of the Sabin-Chumakov collaboration ultimately trumped the ideological differences. Their oral live-virus vaccine became the weapon of choice against polio around the world—including, after its full federal licensing in 1962, for three decades in the U.S. In 1972 Sabin donated his poliovirus strains to the WHO, with the objective of making the vaccine accessible in even the poorest countries.

Today polio remains a serious threat only in parts of Pakistan, Afghanistan and Nigeria. If polio is ever completely eradicated from the globe—as seems more and more possible—the world will have the little-known and improbable collaboration between Albert Sabin and Mikhail Chumakov to thank for it. **SA**

MORE TO EXPLORE

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SCIENTIFIC AMERICAN ONLINE

For a timeline of polio milestones, go to ScientificAmerican.com/apr2012/polio-timeline

PALeontOLOGY

TIME TRAVELER

Artist Charles R. Knight drew on his vast experience depicting living animals to bring prehistoric creatures to life—a practice that made him keenly aware of the finality of extinction

By Richard Milner



SABER-TOOTHED CAT defends its kill from an encroaching *Tarpanus* at the La Brea tar pits in this 1960s painting by Charles R. Knight.



Richard Milner is an associate in the division of anthropology at the American Museum of Natural History in New York City. His latest book is *Charles R. Knight: The Artist Who Saw Through Time* (Abrams, 2012).

YOU MAY NOT KNOW HIS NAME, BUT chances are that you have seen his work. Brooklyn-born artist Charles R. Knight (1874–1953) produced paintings and sculptures of dinosaurs, mammoths and prehistoric humans that adorn the great natural history museums in the U.S. His dinos have appeared as toys, stamps and comics, as well as in books and scientific journals on paleontology. One of Sir Arthur Conan Doyle's illustrators swiped them for his 1912 novel *The Lost World*. Some even became movie stars, directly inspiring sequences in the 1933 *King Kong* and, more indirectly, Walt Disney's 1940 *Fantasia* and Steven Spielberg's 1993 *Jurassic Park*. Hollywood's master monster animator Ray Harryhausen, creator of the dinosaurs in the 1966 *One Million Years B.C.* and other cult classics, based his stop-motion puppets on paintings and sculptures by Knight.

Knight is best known for his depictions of long-extinct beasts, but he was first and foremost a wildlife artist—an underappreciated aspect of his career. Over the course of his lifetime he created nearly 1,000 portraits of living animals representing 800 species—an astonishingly prodigious output. His prehistoric reconstructions benefited from years of keen observations and detailed anatomical studies of modern-day animals. Painting portraits of living lions, tigers, snow leopards and house cats sharpened his portrayal of a snarling saber-toothed cat defending its kill from a giant, condorlike vulture at the La Brea tar pits. Sketches of zoo elephants prepared him to breathe life into woolly mammoths marching across a snowscape in Ice Age France.

In researching my new book *Charles R. Knight: The Artist Who Saw Through Time*, I noticed a previously overlooked subtext in Knight's art and writings. Decades of studying fossil bones with paleontologist Henry Fairfield Osborn, his scientific mentor at the American Museum of Natural History in New York City, impressed Knight with the irrevocability of extinction. He became haunted by the realization that all his beloved animal species were ultimately doomed and that humans were now greatly accelerating the process. During his own lifetime, the once superabundant American bison had been slaughtered



to the brink of extermination. In 1901 the U.S. government belatedly adopted the species as an icon by putting Knight's drawing of a bison bull on a postage stamp and the \$10 bill.

Knight came to regard each living species as an irreplaceable treasure. When individuals became very rare, such as the sole surviving passenger pigeon that died in 1914 at the Cincinnati Zoo, he would hasten to sketch them—an artist's loving homage and farewell. His sympathies did not extend to tyrannosaurs, however. In his 1946 book *Life Through the Ages*, he wrote that the carnosaurs (a group that includes the tyrannosaurs) "have long since vanished, which perhaps is just as well, because no more sinister beings ever walked the surface of this earth."

I was stunned to learn that Knight was practically blind for much of his adult life—an ironic twist of fate for an artist whose images were so influential. He painted small, detailed oil sketches on boards a few inches from his eyes, which assistants meticulously enlarged onto the museum walls. Then he would mount the scaffold to add finishing touches. When he looked up at a completed mural, whether of dueling dinosaurs or giant ground sloths and armadillos, it was all a blur. Yet he persevered.

He wished that people could experience, if only in fantasy, the "lost world" he had visited so often in imagination and proposed a theme park filled with life-size dinosaur statues. Unfortunately, it was never created during his lifetime for lack of a sponsor.

Ten years after his death, however, that dream became a reality, thanks to his friend and collaborator Louis Paul Jonas, a gifted taxidermist and animal sculptor. Jonas raised money from Sinclair Oil and modeled nine lifelike fiberglass dino sculptures, including a 70-foot-long "brontosaurus/apatosaur," for New York's 1964 World's Fair. Thousands flocked to enter this prehistoric world, which was like stepping inside a Knight mural—a fitting memorial for the courageous artist who faced darkness and extinction armed only with clay, plaster and paint. ■

SCIENTIFICAMERICAN ONLINE

For a slide show of Knight's images, go to ScientificAmerican.com/apr2012/knight



CHARLES R. KNIGHT is best known for his pioneering paleoart, which found its way into pop culture. His iconic painting from 1927 of a face-off between a *T. rex* and *Triceratops* (top), for example, later influenced a scene in the cult classic *One Million Years B.C.* But Knight was primarily a wildlife artist, and his depictions of extant creatures, such as this recently discovered drawing of a snow leopard from 1904 (bottom), informed his images of extinct ones. Working on prehistoric animals impressed on Knight the irrevocability of extinction, and he raced to capture the last representatives of species, including the passenger pigeon (opposite page), before they disappeared.

PHOTOGRAPH BY

THE LIMITS OF

BREATH HOLDING

It's logical to think that the brain's need for oxygen is what limits how long people can hold their breath. Logical, but not the whole story

By Michael J. Puplis

Michael J. Parkes is senior lecturer in applied physiology at the School of Sport and Exercise Sciences at the University of Birmingham in England. He also works at the Wellcome Trust Clinical Research Facility at the University Hospitals Birmingham NHS Foundation Trust.



TAKE A DEEP BREATH

and hold it. You are now engaging in a surprisingly mysterious activity. On average, we humans breathe automatically about 12 times per minute, and this respiratory cycle, along with the beating of our heart, is one of our two vital biological rhythms. The brain adjusts the cadence of breathing to our body's needs without our conscious effort. Nevertheless, all of us also have the voluntary ability to deliberately hold our breath for short periods. This skill is advantageous when preventing water or dust from entering our lungs, when stabilizing our chests before muscular exertion and when extending how long we can speak without pause. We hold our breath so naturally and casually that it may come as a surprise to learn that fundamental understanding of this ability still eludes science.

(Feel free to exhale now, if you haven't already.)

Consider one seemingly straightforward question: What determines how long we can hold our breath? Investigating the problem turns out to be quite difficult. Although all mammals can do it, nobody has found a way to persuade laboratory animals to hold their breath voluntarily for more than a few seconds. Consequently, voluntary breath holding can be studied only in humans. If the brain runs out of oxygen during a lengthy session, then unconsciousness, brain damage and death could quickly follow—dangers that would render many potentially informative experiments unethical. Indeed, some landmark studies from past decades are unrepeatable today because they would violate the safety guidelines for human subjects.

Nevertheless, researchers have found ways to begin answering the questions surrounding breath holding. Beyond illuminating human physiology, their discoveries might eventually help save lives both in medicine and in law enforcement.

DETERMINING THE BREAK POINT

IN 1959 physiologist Hermann Rahn of the University at Buffalo School of Medicine used a combination of unusual methods—slowing his metabolism, hyperventilating, filling his lungs with pure ox-

ygen, and more—to hold his breath for almost 14 minutes. Similarly, Edward Schneider, a pioneer of breath-holding research at the Army Technical School of Aviation Medicine at Mitchel Field, N.Y., and, later, Wesleyan University, described a subject lasting for 15 minutes and 13 seconds under comparable conditions in the 1930s.

Still, studies and daily experience suggest that most of us, after inflating our lungs maximally with room air, cannot hold that breath for more than about one minute. Why not longer? The lungs alone should contain enough oxygen to sustain us for about four minutes, yet few people can hold their breath for even close to that long without practice. In the same vein, carbon dioxide (the exhaled waste product made by cells as they consume food and oxygen) does not accumulate to toxic levels in the blood quickly enough to explain the one-minute limit.

When immersed in water, people can hold their breath even longer. This extension may stem in part from increased motivation to avoid flooding the lungs with water (it is unclear whether humans possess the classical diving reflex of aquatic mammals and birds that lowers their metabolic rate during breath holding while submerged). But the principle remains true: breath-holding divers feel compelled to draw a breath well before they actually run out of oxygen.

IN BRIEF

What determines how long someone can hold a breath? People usually need to gasp for air long before their brain or body runs out of oxygen (the obvious limitation).

Investigating what limits our control over breath

holding has been difficult, but decades of research suggest that the diaphragm, which contracts to inflate the lungs, plays a key role.

The best hypothesis is that the diaphragm sends signals to the brain about how long it has been con-

tracted and how it is biochemically reacting to depleted levels of oxygen or rising levels of carbon dioxide. Initially those signals cause mere discomfort, but eventually the brain finds them intolerable and forces breathing to start again.

As Schneider observed, "it is practically impossible for a man at sea level to voluntarily hold his breath until he becomes unconscious."• Unconsciousness might occasionally occur under unusual circumstances, such as in extreme diving competitions, and some anecdotes suggest rare cases in which children can hold their breath long enough to pass out, but laboratory studies confirm that normally we adult humans cannot do it. Long before too little oxygen or too much carbon dioxide can hurt the brain, something apparently brings us to the break point (as researchers call it) past which we cannot resist gasping for air.

One logical, hypothetical explanation for the break point is that specialized sensors in the body observe physiological changes associated with breath holding and trigger a breath before the brain shuts down. Obvious candidates for such sensors would be ones that watched for lengthy expansions of the lungs and chest or that detected reduced levels of oxygen or elevated levels of carbon dioxide in the blood or the brain. Neither of those ideas appears to hold up, however. The involvement of volume sensors in the lungs appears to have been ruled out by various experiments conducted between the 1960s and the 1990s by Helen R. Harty and John H. Eisele, working independently in Abe Guz's laboratory at Charing Cross Hospital in London, and by Patrick A. Flume, then at the University of North Carolina at Chapel Hill. Their experiments showed that neither lung-transplant patients, whose nerve connections between lungs and brain were severed, nor patients receiving complete spinal anesthesia, whose chest-muscle sensory receptors were blocked, could hold their breath for abnormally long periods. (It is significant that those anesthesia experiments did not affect the diaphragm muscle, however, for reasons that will become apparent.)

Research also seems to exclude the involvement of all the known chemical sensors (chemoreceptors) for oxygen and carbon dioxide. In humans, the only known sensors detecting low blood oxygen levels are in the carotid arteries just underneath the angle of the jaw, which supply blood to the brain. The chemoreceptors detecting raised carbon dioxide levels are in the carotid arteries and in the brain stem, which controls regular breathing and the other autonomic (involuntary) functions.

If the oxygen chemoreceptors caused the urgent sensation of break point, then without their feedback, people ought to be able to hold their breath until rendered unconscious. Experiments in Karlman Wasserman's laboratory at the University of California, Los Angeles, have shown, however, that patients still cannot do so if the nerve connections between chemoreceptors in their carotid arteries and the brain stem are severed.

What Triggers Break Point?

Break point is the moment during a held breath when it becomes impossible for the breath holder to resist gasping for air. Training in breath holding can extend it, as can meditation, flooding the body with oxygen and purging it of carbon dioxide (CO₂). Finding what truly determines break point has nonetheless been frustratingly difficult. Research has ruled out some possibilities, however, and the beginnings of an explanation might be in sight.

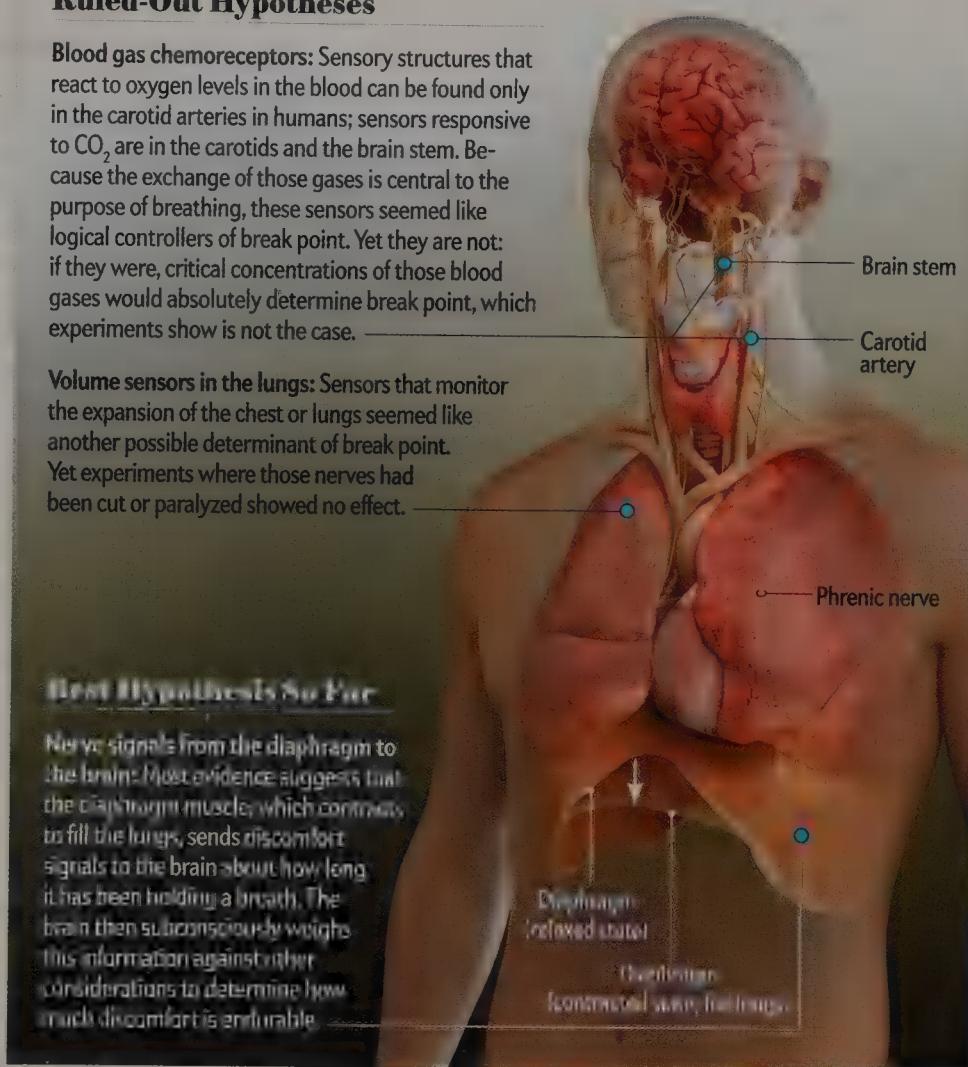
Ruled-Out Hypotheses

Blood gas chemoreceptors: Sensory structures that react to oxygen levels in the blood can be found only in the carotid arteries in humans; sensors responsive to CO₂ are in the carotids and the brain stem. Because the exchange of those gases is central to the purpose of breathing, these sensors seemed like logical controllers of break point. Yet they are not: if they were, critical concentrations of those blood gases would absolutely determine break point, which experiments show is not the case.

Volume sensors in the lungs: Sensors that monitor the expansion of the chest or lungs seemed like another possible determinant of break point. Yet experiments where those nerves had been cut or paralyzed showed no effect.

Best Hypothesis So Far

Nerve signals from the diaphragm to the brain: Most evidence suggests that the diaphragm muscle, which contracts to fill the lungs, sends discomfort signals to the brain about how long it has been holding a breath. The brain then subconsciously weighs this information against other considerations to determine how much discomfort is endurable.



Moreover, if reduced oxygen or elevated carbon dioxide levels alone dictated the break point, then beyond some threshold levels, breath holding should be impossible. Yet numerous studies have shown this not to be the case. It would also be true that after the gas levels triggered a break point, breath holding would remain impossible until the arterial oxygen and carbon dioxide levels returned to normal. But that prediction is not borne out, either, as researchers have casually observed since the early 1900s. In 1954 Ward S. Fowler of the Mayo Clinic described formally how after maximum breath holding, subjects could immediately do it a second time if they inhaled only an asphyxiating gas—and even a third time, despite their blood gas levels becoming progressively worse.

Further work has verified that this remarkable repeated breath-holding capability is independent of the number or vol-

ume of inhalations of the asphyxiating gas. Indeed, in 1974 John R. Rigg and Moran Campbell, both at McMaster University in Ontario, demonstrated that it persists even when the subjects merely attempt to exhale and inhale with their airway closed.

Taken together, all these experiments involving repeated breath-holding maneuvers suggest that the need to draw a breath somehow relates to the muscular act itself and not directly to its gas-exchange functions. When the chest is greatly inflated, its natural tendency is to recoil unless the inspiratory muscles of breathing hold it in the inflated state. So researchers of the break point began to look for answers in the body's neurological and mechanical controls over these inspiratory breathing muscles. As part of that work, they also wanted to learn whether breath holding involves a voluntary halt of the automatic breath-

ing rhythm that drives these muscles or the prevention of the breathing muscles from expressing this automatic rhythm.

UNREPEATABLE EXPERIMENTS

THE NORMAL RHYTHM of our breathing can be said to begin when the brain stem sends impulses down our two phrenic nerves to the bowl-shaped diaphragm muscle underneath the lungs, telling it to contract and inflate the lungs. When the impulses stop, the diaphragm relaxes and the lungs deflate. In other words, some rhythmic pattern of neural activity—a central respiratory rhythm—mirrors the cycle of our breaths. In humans it is still technically and ethically impossible to measure this central rhythm directly from the phrenic nerves or from the brain stem. Investigators have devised ways to record the central respiratory rhythm indirectly, however: by monitoring instead the electrical activity in the diaphragm muscle, the pressure in the airway or other changes in the autonomic nervous system, such as the heartbeat rhythm (known as respiratory sinus arrhythmia).

Working from such indirect measurements, Emilio Agostoni of the University of Milan in Italy showed in 1963 that he could detect a central respiratory rhythm in human subjects holding their breath well before they reached break point. In related experiments at the University of Birmingham in England in 2003 and 2004, graduate student Hannah E. Cooper, anesthetist Thomas H. Clutton-Brock and I used respiratory sinus arrhythmia to show that the central respiratory rhythm never stops: it persists throughout breath holding. Breath holding must therefore involve suppressing the diaphragm's expression of this rhythm, possibly through a voluntary, continuous contraction of that muscle. (Various experiments seem to have ruled out the involvement of other muscles and structures involved in normal breathing.) Break point may similarly depend on sensory feedback to the brain from the diaphragm—reflecting, for example, how stretched or unusually overworked it may be.

If so, then paralyzing the diaphragm to eliminate its sensory feedback to the brain ought to allow subjects to prolong their breath holding greatly if not indefinitely. Such was the expectation in one of the most alarming breath-holding experiments ever, which Campbell performed at Hammersmith Hospital in London in the late 1960s. Two healthy, conscious volunteers consented to have all their skeletal muscles temporarily paralyzed with intravenous curare—except for one forearm, with which they could signal their wishes. The subjects were kept alive with a mechanical ventilator; breath holding was simulated by switching it off, and the subjects indicated their break point by signaling when they wanted the ventilator restarted.

The result was astonishing. Both volunteers were happy to leave the ventilator switched off for at least four minutes, at which point the supervising anesthetist intervened because their blood carbon dioxide levels had risen perilously. After the effects of the curare had worn off, both subjects reported feeling no distressing symptoms of suffocation or discomfort.

For obvious reasons, such a daring experiment has rarely been repeated. Some others have tried and failed to replicate Campbell's findings, but their courageous volunteers reached break point after such a short duration that their carbon dioxide levels barely rose above normal. Those observations suggest that the subjects might have chosen to end the tests early, possibly because of discomfort from the air tubes holding open the

A REALM OF THEIR OWN

Secrets of Champions

People who excel at breath holding often rely on four key principles. Extended breath holding poses serious risks for unconsciousness, brain injury and death, however. Medical assistance should always be standing by.

REALLY FILL THE LUNGS: Some athletes hyperinflate the lungs beyond their normal maximum through a technique known as buccal pumping, rhythmically moving the floor of the mouth to draw in extra air. The elevated pressures inside the lungs that result, however, pose a risk of arterial gas embolism—gas bubbles in the blood that can damage the brain or coronary capillaries.

RELAX TO SLOW METABOLISM: At rest, human metabolism consumes about 0.36 liter of oxygen per minute. By fasting for 12 hours and lying quietly awake, one can lower oxygen consumption to just 0.27 liter per minute, which makes the air in the lungs last about 33 percent longer.

INHALE PURE OXYGEN: Fresh air is usually only about 21 percent oxygen. Studies show that inhaling 100 percent oxygen can double the duration of breath holding. Yet doing so also raises the possible danger that regions of the lungs may collapse once all the oxygen they contain is extracted.

HYPERVENTILATE: Hyperventilation before breath holding can lower the levels of carbon dioxide in the blood, which in studies has sometimes doubled the time until break point. Yet it can also be counterproductive: hyperventilation tends to speed up how quickly the body consumes oxygen and produces carbon dioxide. Moreover, it restricts the supply of blood reaching the brain and disarms reflexes that protect the brain from inadequate oxygen. —The Editors

Notable Records*



*Achieved while motionless and facedown in water, without first inhaling pure oxygen

glottis (a modern safety requirement not present in Campbell's experiment) and because of their greater awareness of the life-threatening risk. Nevertheless, some equally remarkable experiments by Mark I. M. Noble, working in Guz's laboratory at Charing Cross Hospital in the 1970s, seem to confirm that diaphragm paralysis prolongs breath-holding duration. Instead of total body paralysis, Noble and his colleagues used the much less life-threatening maneuver of paralyzing the diaphragm alone by anesthetizing only the two phrenic nerves. Doing so doubled subjects' average breath-holding duration and reduced the usual uncomfortable sensations that accompany breath holding.

CURRENT BEST EXPLANATION

THE BALANCE OF EVIDENCE thus favors the speculation that a voluntary, lengthy contraction of the diaphragm holds the breath by keeping the chest inflated. The break point may depend very much on stimuli that reach the brain from the diaphragm in this unusual contracted state. During such a lengthy contraction, the brain might subconsciously perceive the unusual signals from the diaphragm as vaguely uncomfortable at first but eventually as intolerable, causing the break point. The automatic rhythm then regains control.

This hypothesis is not fully fleshed out, but it fits nicely both with Fowler's observations (that any release of breath holding, necessarily by relaxing the diaphragm, enabled another one) and with the effects of lung inflation and blood-gas manipulation on breath-holding duration. Relaxing the diaphragm even a bit and exhaling slightly would delay break point by relieving the signals from the stretch sensors in the diaphragm. Raising the oxygen level and lowering the carbon dioxide level in the blood would also extend breath-holding capability by reducing biochemical indicators of fatigue in the diaphragm. Anything that prevents the brain from monitoring such information—for example, by blocking the nerves between the diaphragm and the brain—will extend duration. The tolerance of the brain to such unpleasant signals will also depend on your mood, motivation and ability to be distracted by, say, mental arithmetic.

This hypothesis is only the simplest unifying explanation for the experimental observations. Some of these experiments used too few subjects to be the basis for reliable generalizations, and ethical permission to repeat them may never be granted. Key pieces of the jigsaw puzzle may still be missing.

Moreover, a puzzle piece that does not yet quite fit comes from another of Noble and Guz's dramatic (and now ethically unrepeatable) breath-holding experiments. They tripled the duration of breath holding in three healthy subjects by anesthetizing their two sets of cranial nerves (the vagus nerves, which go from the brain to organs in the chest and abdomen, and the glossopharyngeal nerves, which go to the glottis, larynx and other parts of the throat). This result would appear to have been achieved without affecting the diaphragm, except that it is also possible that the vagus nerves, too, carry some signals from the diaphragm. It seems less likely that the larynx itself contains a muscle involved in breath holding: in 1993 when surgeon Martyn Mendelsohn of Sydney, Australia, viewed the glottis (via a camera inserted through a nostril), the glottis often remained open throughout breath holding. This observation, too, seems to support the conjecture that the diaphragm's role is key.

SAVING LIVES

BETTER UNDERSTANDING of what limits people's ability to hold their breath has practical uses in medicine. As part of the treatment for breast cancer, for instance, patients receive radiation therapy, during which the goal is to lethally dose the entire tumor without damaging the healthy tissues all around it. Doing so requires minutes of radiation exposure, during which a patient must try to keep her breast motionless. Because breath holding for so long is impractical, current practice uses short bursts of radiation timed to fall between a patient's breaths, when her chest is moving least. Yet with each breath, the breast moves and may not necessarily return to exactly the same position. Medical physicist Stuart Green, clinical oncologist Andrea Stevens, anesthetist Clutton-Brock and I are now starting experiments funded by University Hospital Birmingham Charities to test whether it would be feasible to prolong breath holding sufficiently to aid radiotherapy treatment.

A practical understanding of breath holding might also be of value to law-enforcement personnel when they are forcibly restraining suspects. Every year around the world some people under restraint may die accidentally. Raising the metabolic rate, compressing the chest, lowering the blood oxygen level and raising the blood carbon dioxide level all shorten the duration of a person's breath holding. So someone who is angry, has been fighting or is being forcibly held down may well need to draw a breath earlier than someone who is relaxed.

In 2000 Andrew R. Cummin and his team at Charing Cross Hospital studied what happened after eight healthy subjects breathed out maximally and held their breath after cycling moderately for one minute: the duration of their maximum breath holding plummeted to 15 seconds, the average amount of oxygen in their blood fell dramatically and two of them developed irregular heartbeats. Consequently, the researchers concluded that the "cessation of breathing for short periods during vigorous restraint ... may account for unexplained deaths in these circumstances." Law-enforcement authorities have carefully compiled guidelines for the use of forcible restraint; they should be observed scrupulously.

Such investigations of breath holding open windows into vital aspects of human physiology. Clearly, more groundbreaking discoveries, particularly about the diaphragm itself, remain ahead—which leaves some of us breathless in anticipation. ■

MORE TO EXPLORE

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Behavioural and Arousal-Related Influences on Breathing in Humans. S. A. Shea in *Experimental Physiology*, Vol. 81, No. 1, pages 1–26; 1996.

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SCIENTIFIC AMERICAN ONLINE

Learn how the diving reflex may extend breath holding underwater at ScientificAmerican.com/apr2012/breath-holding

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NUCLEAR ASTROPHYSICS

Speaker: David Lunney, Ph.D.

■ *Author's Guide to the Universe*

An introduction to the formation and composition of the visible universe, emphasizing the synthesis of Earth's chemical elements in the stars. Discover the key reactions, the evolutionary process of nuclear systems, and the forces that shape ongoing debates in nuclear astrophysics.

Nuclear Cooking Class

Get cooking with a discussion of the physics behind element formation by fusion and capture reactions. Dr. Lunney will highlight the need to weigh ingredient atoms to precisely determine mass. Take a seat in a precise corner of the physics kitchen and feast on the latest on nucleosynthesis.

Weighing Single Atoms

The most precise balance known to man is an electromagnetic trap in which ionized atoms are made to dance, revealing their mass. We'll look at the basics of atomic mass measurement. Learn about current techniques of mass measurement, how these methods compare, and the diverse programs worldwide that use them. Glimpse the shape of the future of precision measurement.

Panning for Seafloor for Plutonium: of the Sun

Long, long ago, not so far away, did an exploding supernova bathe our planet with its stellar innards? Explore the research, theories, and phenomena that suggest the role of a local supernova in the creation of the sun and its planetary system.



NEUROSCIENCE MEMORY

Speaker: Jeanette Norden, Ph.D.

How the Brain Works

Get the lay of the land in this introductory neuroscience session showing how the brain is divided into functional systems. A special emphasis will be on limbic and reticular systems, which underlie learning and memory, executive function, arousal, attention, and consciousness.

Memory and All That Jazz

Memory is among the most precious of human abilities. Find out what neuroscience has revealed about how we learn and remember. Pinpoint how different areas of the brain encode different types of information—from the phone number we need to remember for only a moment to the childhood memories we retain for a lifetime.

Losing your Memory

When we lose our memories, we lose a critical part of ourselves and our lives. Dr. Norden will introduce the many clinical conditions that can affect different types of learning and memory.

Don't or Lose It!

While memory can be lost under a wide variety of clinical conditions, most memory loss during aging is not due to strokes or neurodegenerative disease, but to lifestyle. Building evidence suggests that aging need not lead to significant memory loss. Find out how to keep your brain healthy as you age.



COGNITIVE NEUROSCIENCE

Speakers: Stephen Macknik, Ph.D. and Susana Martinez-Conde, Ph.D.

How the Brain Constructs the World We See

All understanding of life experiences is derived from brain processes, not necessarily the result of actual events. Neuroscientists are researching the cerebral processes underlying perception to understand our experience of the universe. Discover how the brain constructs, not reconstructs, the world we see.





Cognitive Neuroscience, cont.

Windows on the Mind

What's the connection behind eye movements and subliminal thought? Join Dr. Macknik and Dr. Martinez-Conde in a look at the latest neurobiology behind microsaccades, the involuntary eye movements that relate to perception and cognition. Learn how microsaccades suggest bias toward certain objects, their relationship to visual illusions, and the pressing questions spurring visual neurophysiologists onward.

Champions of Illusion

The study of visual illusions is critical to understanding the basic mechanisms of sensory perception and advancing cures for visual and neurological diseases. Connoisseurs of illusion, Dr. Macknik and Dr. Martinez-Conde produce the annual Best Illusion of the Year Contest. Study the most exciting novel illusions with them and learn what makes these brain tricks work.

Slights of Mind

Magic fools us because humans have hardwired processes of attention and awareness that can be "hacked." A good magician employs the mind's own intrinsic properties. Magicians' insights, gained over centuries of informal experimentation, have led to new discoveries in the cognitive sciences, and reveal how our brains work in everyday situations. Get a front-row seat as the key connections between magic and the mind are unveiled!



CLIMATOLOGY

Speaker: Yohay Carmel, Ph.D.

Prioritizing Land for Nature

Conservation: Theory and Practice

Forest clearing, climate change, and urban sprawl are transforming our planet at an accelerating rate. Conservation planning prescribes principles and practical solutions for selecting land for protection, assigning land for development, and minimizing the negative impact on nature. Taking a bird's-eye view of approaches to conservation, we'll put the hot topics and tough questions in perspective through an insightful discussion.

Facing ■ ■ ■ Mega-Fire Reality

Worldwide, the area, number, and intensity of wildland fires has grown significantly in the past decade. Fire-protection strategies used in the past may not work in the future. Learn the roots and causes of wildfires and recent efforts to predict, manage, and mitigate fire risk. Gain food for thought about the complex interface between science and policy.

HUMAN EVOLUTION

Speaker: Chris Stringer, Ph.D.

Human Evolution: The Big Picture

Time-travel through 6 million years of human evolution, from the divergence from African apes to the emergence of humans. In 1871, Charles Darwin suggested that human evolution had begun in Africa. Learn how Darwin's ideas stand up to the latest discoveries, putting his tenets into context and perspective.

The First Humans

About 2 million years ago the first humans appeared in Africa, distinctly different from their more ancient African ancestors. Discover what drove their evolution and led to a spread from their evolutionary homeland to Asia and Europe. Explore current thinking on the early stages of human evolution.

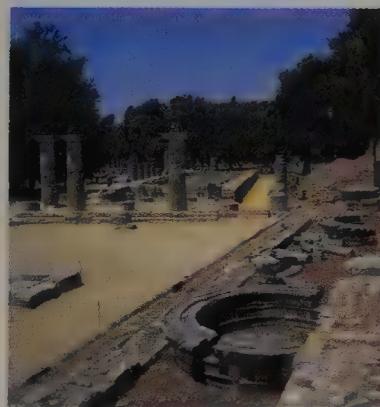
The Neanderthals

Another Kind of Human

Our close relatives, the Neanderthals, evolved in parallel with *Homo sapiens*. Often depicted as bestial ape-men, in reality they walked upright as well as we do, and their brains were as large as ours. So how much like us were they? What was their fate? Track the evolution of the Neanderthals in light of the latest discoveries.

The Rise of *Homo Sapiens*

Modern humans are characterized by large brains and creativity. How did our species arise and spread across the world? How did we interact with other human species? We will examine theories about modern human origins, including Recent African Origin ("Out of Africa"), Assimilation, and Multiregional Evolution, and delve into the origins of human behavioral traits.



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Whether you lean toward concept or application, there's much to pique your curiosity. Discover the excitement of fundamental research and get an insider's look at the world's largest particle physics laboratory.

Our full-day tour will be led by a CERN physicist. We'll have an orientation, visit an accelerator and experiment, get a sense of the mechanics of the Large Hadron Collider (LHC), make a refueling stop for lunch, and have time to peruse exhibits and media on the history of CERN and the nature of its work.

This tour includes: Bus transfer from Geneva, Switzerland to our Genoa, Italy hotel (October 23) • 3 nights' hotel (October 20, 21, 22) • 3 full breakfasts (October 21, 22, 23) • Transfers to and from the hotel on tour day (October 22) • Lunch at CERN • Cocktail party following our CERN visit • Do-as-you-please day in Geneva, including transfers to and from downtown (October 21) • Transfer from airport to our Geneva hotel

The price is \$899 per person (based on double occupancy). This trip is limited to 50 people. NOTE: CERN charges no entrance fee to visitors.



EPHESUS

November 1, 2012—

Many civilizations have left their mark at Ephesus. It's a complex and many-splendored history, often oversimplified. Bright Horizons pulls together three important aspects of understanding Ephesus that are rarely presented together. You'll meander the Marble Road, visit the legendary latrines, check out the Library, and visit the political and commercial centers of the city. A visit to the Terrace Houses will enhance your picture of Roman-era Ephesus.

We'll take a break for Mediterranean cuisine in the Selcuk countryside, then visit the Ephesus Museum in Selcuk, where city excavation finds are showcased, and you'll get a fuller look at local history, from the Lydians to the Byzantines.

ATHENS

November 1, 2012—

The Parthenon and its Acropolis setting are stunning, no doubt about it. Requiring no interpretation, they are ideal for a DIY Athens excursion. On the other hand, visiting the new Acropolis Museum and the National Archaeological Museum with a skilled guide who's on your wavelength adds immeasurably to the experience. We suggest you join Bright Horizons on a focused trip. You'll see the Parthenon frieze, exquisite sanctuary relics, and Archaic sculpture at the Acropolis Museum (as you can see from the picture, the museum sits just below the Acropolis).



Lunch is tucked away at a taverna favored by Athenian families. For dessert, we'll visit the richest array of Greek antiquities anywhere—at the National Archaeological Museum.

APRIL 11TH – 25TH, 2013

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★ www.InsightCruises.com/sciam17

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MOLECULAR BIOLOGY

Speaker: John Mattick, Ph.D.

The History of Molecular Biology and the New Age of Medicine

The 20th century was dominated by nuclear physics, organic chemistry, electronics and computing. It also saw the birth of molecular biology, which came of age with the 2001 sequencing of the human genome, at a cost of \$3 million. Today we can sequence a human genome for just \$3,000, and the price is dropping quickly. Come hear about the events leading up to the founding of molecular biology and the human genome project and how genomics is set to revolutionize society, medicine, and your health future.

The Programming of Human Development

The human genome is a marvel. It is a zip file containing 3 billion letters (equivalent to 6 gigabytes) that programs the development of an organism with 100 trillion cells arranged into a myriad of muscles, bones, and organs. Less than 2% of the human genome encodes proteins, with most of the rest thought to be junk because it does not fit the conventional concept of a gene. Find out how molecular biology now understands the structure of human genetic programming.

The Evolution and Molecular Basis of Human Cognition

Life has existed on Earth for around 4 billion years, mostly in microbial form. It was not until the last billion years that developmentally complex organisms began to evolve, culminating 500 million years ago in the so-called 'Cambrian explosion,' when ancestors of all animal phyla appeared. While plants and animals diversified, information processing capacity evolved which ultimately led to the rise of human intelligence and cognition. Come hear about the developments which led to the evolution of intelligence in the primates.

Epigenetic Inheritance

For decades it has been an article of faith in evolutionary biology that mutation is random and that sperm and ova are immune from environmental influence. However, recent evidence suggests that the processes that control our

development can be influenced by experience and that experience can be transmitted between generations. Learn how we may be the product of both our ancestors' genes and their experiences, and how the new field of 'epigenetics' is influencing ideas of health and health policy.



COSMOLOGY

Speaker: Elaine Sadler, Ph.D.

The Lives of Galaxies

How did galaxies like our own Milky Way begin? Why do galaxies look the way they do, and how do they change over cosmic time? Learn the latest ideas and findings on both nearby and distant galaxies as well as how our understanding of galaxies as 'cosmic ecosystems' has progressed rapidly in recent years.

Secrets of the Invisible Universe

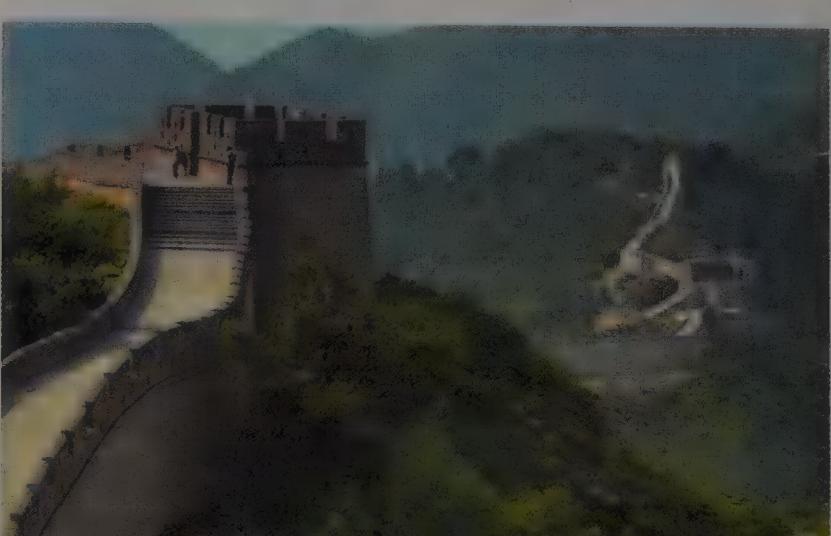
Modern telescopes allow us to study the universe at radio, X-ray and gamma-ray wavelengths which are invisible to the human eye. They reveal an energetic and sometimes violent universe populated by supermassive black holes, exploding stars and other exotic cosmic phenomena. Come discover some of the many secrets of the invisible universe.

Astronomy in Australia

Astronomy is seen as a flagship of Australian science. Find out why in a short history of Australian astronomy from the Dreamtime to the present day. You'll learn about some of the main astronomical observatories and instruments in Australia, including those currently under construction as pathfinders for the international Square Kilometre Array (SKA) radio telescope.

Serendipity and Discovery in Astronomical Astronomy

All scientists hope to make discoveries which change our view of the world, and the past 100 years have seen immense shifts in our understanding of the Universe we live in. So how is progress really made? We will discuss some Nobel-prizewinning discoveries, the search for exoplanets, the role of serendipity in astronomical discovery, the contributions of amateur astronomers and the rise of 'citizen science'.





PLANETARY SCIENCE

Speaker: Stephen P. Maran, Ph.D.

To Hubble and Beyond

How do Galileo's mind-blowing first telescopic discoveries contrast with current knowledge of the same celestial phenomena, examined with 21st century telescopes and space probes? Both Galileo and Hubble Space Telescope focus on centers of revolution, moons, planets, and rings, and galaxies. Find out how 17th and 21st century optical astronomy compare and relate.

Mystery Forces in the Solar System

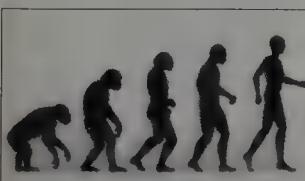
Astronomers have investigated puzzles and discrepancies noted in the paths of moving bodies, and discovered previously unknown celestial objects and astrophysical phenomena. While each mystery solved is just a footnote in space discovery, together they demonstrate the unforeseen benefits of scientific exploration. Get the details with Stephen Maran.

Through Time and Space with the Hubble Space Telescope

What is the significance of the Hubble Space Telescope? Join Dr. Maran for a look at the whats and hows, highs and lows of the Hubble Space Telescope. The epic story spans vision, disaster, innovation, and outstanding discovery, much of which was unforeseen when the Hubble project began. Listen in on missions accomplished and new beginnings afoot.

Exoplanets, Life in Space

My, how things have changed! For years astronomers largely denied the existence of exoplanets. Now astronomers find planets wherever they look. Explore the stunning contributions of NASA's planet-hunting Kepler mission to the search for exoplanets and Goldilocks zones where life could exist. Join the discussion about the possibilities and implications.



EVOLUTION

Speaker: Mohamed Noor, Ph.D.

What is "Evolution" Anyway and Why Should I Care?

The mere word "evolution" conjures images in the public ranging from movie dinosaurs to something vaguely half-human-half-gorilla. What does the word "evolution" actually mean in the biological sciences, what is the evidence that it is "true", and why should the general public know and care? Evolution affects your everyday life, from your health to your livelihood, and this class will help you learn why!

Sexual Selection — Trials and Tribulations of Picking a Mate

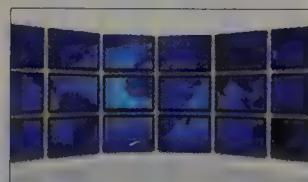
Darwin recognized the importance of "sexual selection" in causing elaborate displays and differences between species. Recent work, however, shows that adaptations to get more matings or offspring sometimes causes harm to the other sex, and results in "arms races" between males and females. Come hear and see some interesting case studies on the trials and tribulations of picking (or resisting) a mate.

Molecular Adaptation

While we know some traits are "adaptive," like giraffe's necks or "bad" like genetic diseases, what is the genetic basis of these traits and what evolutionary forces affect them? With the growth of genetic mapping and the emergence of extensive genome sequencing, we have far more case studies of "molecular adaptation" than ever in the past. We will explore classic and recent advances in the study of molecular adaptation.

The Mania of Recombination

Geneticists often describe "mutations" as the ultimate source of all genetic variation. However, genetic recombination is fundamentally important in all realms of genetics and evolution—ranging from the evolution of sex to the formation of new species to generating variation on which natural selection can act. Come learn some of the evolutionary wonder associated with this basic genetic process.



GEOSPATIAL IMAGING

Speaker: Murray Felsher, Ph.D.

Observing a Changing World

Geospatial imaging uses an array of remote sensing technologies to image the Earth from Space. Gain a basic understanding of how sensor technology now aboard earth-orbiting spacecraft provides data and information about planet Earth. Join Dr. Felsher in a program which will test your assumptions, expand your horizons, and pique your curiosity.

Topics include:

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- Environmental applications: desertification and deforestation and oil spills
- Science applications: meteorology, oceanography, and hydrology
- Policy and political considerations: land use planning, coastal zone management
- Homeland defense and security implications
- "The View From Space: Planet Earth as an Artist's Palette", a look at terrestrial images from an aesthetic perspective

GUILIN, HONG KONG, AND MACAU

Pre-Cruise: April 8–12

— While locals assert that Guilin's mountain and water scenery is "best under heaven" any visitor can agree that the region's otherworldly beauty is among the most picturesque anywhere. Soak in Guilin's vistas, then contrast it with the bustle and urban delights of Hong Kong, China's longtime interface with the Western world.



Trip Includes: • Visits to Guilin Village, Barrier Gate, A-Ma Temple, and St. Paul's Cathedral • 3 nights accommodation in outstanding hotels (Shangri-la Hotel in Guilin, Ritz Carlton Hotel in Hong Kong) • Services of a Western bilingual China Host and local tour guides • All land transportation (as listed on the itinerary) • Entrance fees to all tourist sites (as listed on the itinerary)



BEIJING

Mid-Cruise: April 20–22—Simply put, Imperial Tours has made a connoisseur's visit to Beijing possible. Extraordinary access to behind the scenes, extraordinary experiences, and extraordinary memories await you.

Trip Includes: • Visits to the Great Wall, Tian'anmen Square, the Forbidden Palace, and the Summer Palace • 2 nights accommodation in an outstanding five star hotel (China World Summit Wing) • Services of a Western bilingual China Host and local tour guides • All land transportation (as listed on the itinerary) • Entrance fees to all tourist sites (as listed on the itinerary)

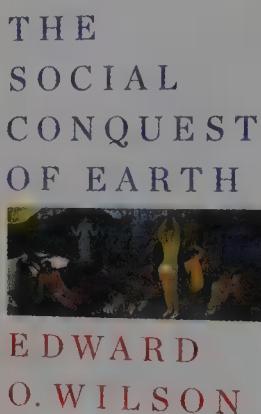
SHANGHAI & XI'AN

Post-Cruise: April 25–28 — Tap into 2,300 years of China's vitality, continuity, and beauty. From Shanghai's dynamism to Xi'an's Silk Road heritage and incomparable terra cotta warriors, you'll sense China's rich, complex history.

Trip Includes: • Visits to Yu Garden, Terracotta Warriors, and the Great Mosque in Xi'an • 3 nights accommodation in outstanding five star hotels (Peninsula Hotel Shanghai and Hilton Xi'an) • Services of a Western bilingual China Host and local tour guides • All land transportation (as listed on the itinerary) • Entrance fees to all tourist sites (as listed on the itinerary)



BOOKS



The Social Conquest of Earth

by Edward O. Wilson. Liveright, 2012 (\$27.95)

The Harvard University naturalist and Pulitzer Prize winner angered many colleagues two years ago, when he repudiated a concept within evolutionary theory that he had brought to prominence. Known as kin selection or inclusive fitness, the half-century-old idea helped to explain the puzzling existence of altruism among animals. Why, for instance, do some birds help their parents raise chicks instead of having chicks of their own? Why are worker ants sterile? The answer, according to kin selection theory, has been that aiding your relatives can sometimes spread your common genes faster than bearing offspring of your own.

In *The Social Conquest of Earth*, Wilson offers a full explanation of his latest thinking on evolution. Group dynamics, not selfish genes, drive altruism, he argues: "Colonies of cheaters lose to colonies of cooperators." As the cooperative colonies dominate and multiply, so

do their alleged "altruism" genes. Wilson uses what he calls "multilevel selection"—group and individual selection combined—to discuss the emergence of the creative arts and humanities, morality, religion, language and the very nature of humans. Along the way, he pauses to reject religion, decry the way humans have despoiled the environment and, in something of a non sequitur, dismiss the need for manned space exploration. The book is bound to stir controversy on these and other subjects for years to come.



by Michel Mitov. Translated by Giselle Weiss. Harvard University Press, 2012 (\$22.95)

A slim, engaging volume that mixes mini lessons on such subjects as thixotropic fluids—think house paint and ballpoint pen ink, both of which flow when someone applies pressure to them but gel when left alone—with anecdotes from the author's adventurous life. In one, Mitov, a liquid-crystal expert in France, travels to Naples, Italy, to solve the mystery behind a religious ritual: why a vial of dried "blood" associated with the martyr San Gennaro often liquefies when brought near the saint's relics. Although Mitov fails to find a definite answer, he concludes it must be a "yield-stress fluid" that changes with time, temperature and

touch. Readers come away from the book with a renewed appreciation for the complexity of such everyday substances as champagne, rubber and toothpaste.



Consciousness: Confessions of a Romantic Reductionist
by Christof Koch.
MIT Press, 2012 (\$24.95)

Neuroscientist Koch first stumbled across Francis Crick, the preeminent molecular biologist, lounging under an apple tree in Germany. A few years later the two launched a decades-long inquiry into the problem of consciousness. The hunt for the essential substrate of our every thought propels Koch through a whirlwind tour of neuroscience, philosophy, physics and information science. The power of the mind's "zombie routines"—those neural machinations, underneath our awareness, that drive a startling number of our decisions and actions—further underscores the mystery of consciousness. We may be less free than we think, but Koch clings to the belief that we are still the masters of our own lives.

—Sandra Upson



BY THE NUMBERS

10¹⁶

Rough number of all the ants living on earth: 10,000 trillion. Combined, they weigh about as much as all the humans.

SOURCE: *The Social Conquest of Earth*

EXHIBITS

Space Shuttle Discovery. Smithsonian National Air and Space Museum, Washington, D.C. The former workhorse of the U.S. space fleet (right) is due to arrive at the museum's Udvar-Hazy Center near Dulles International Airport on April 19. Check the museum's Web site for updates: <http://airandspace.si.edu>

Creatures of Light: Nature's Bioluminescence. American Museum of Natural History, New York City. Open until January 6, 2013. Visitors learn about the nature of bioluminescence in mushrooms, fireflies and sea creatures and the ways scientists study the phenomenon.

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Climbing Mount Immortality

How awareness of our mortality may be a major driver of civilization

Imagine yourself dead. What picture comes to mind? Your funeral with a casket surrounded by family and friends? Complete darkness and void? In either case, you are still conscious and observing the scene. In reality, you can no more envision what it is like to be dead than you can visualize yourself before you were born. Death is cognitively nonexistent, and yet we know it is real because every one of the 100 billion people who lived before us is gone. As Christopher Hitchens told an audience I was in shortly before his death, “I’m dying, but so are all of you.” Reality check.

In his book *Immortality: The Quest to Live Forever and How It Drives Civilization* (Crown, 2012), British philosopher and *Financial Times* essayist Stephen Cave calls this the Mortality Paradox. “Death therefore presents itself as both inevitable and impossible,” Cave suggests. We see it all around us, and yet “it involves the end of consciousness, and we cannot consciously simulate what it is like to not be conscious.”

The attempt to resolve the paradox has led to four immortality narratives: Staying alive: “Like all living systems, we strive to avoid death. The dream of doing so forever—physically, in this world—is the most basic of immortality narratives.” Resurrection: “The belief that, although we must physically die, nonetheless we can physically rise again with the bodies we knew in life.”

Michael Shermer is publisher of *Skeptic* magazine (www.skeptic.com). His new book is *The Believing Brain*. Follow him on Twitter @michaelshermer



Soul: The “dream of surviving as some kind of spiritual entity.” Legacy: “More indirect ways of extending ourselves into the future” such as glory, reputation, historical impact or children.

All four fail to deliver everlasting life. Science is nowhere near reengineering the body to stay alive beyond 120 years. Both religious and scientific forms of resurrecting your body succumb to the Transformation Problem (how could you be reassembled just as you were and yet this time be invulnerable to disease and death?) and the Duplication Problem (how would duplicates be different from twins?). “Even if DigiGod made a perfect copy of you at the end of time,” Cave conjectures, “it would be exactly that: a copy, an entirely new person who just happened to have the same memories and beliefs as you.” The soul hypothesis has been slain by neuroscience showing that the mind (consciousness, memory and personality patterns representing “you”) cannot exist without the brain. When the brain dies of injury, stroke, dementia or Alzheimer’s, the mind dies with it. No brain, no mind; no body, no soul.

That leaves us with the legacy narrative, of which Woody Allen quipped: “I don’t want to achieve immortality through my work; I want to achieve it by not dying.” Nevertheless, Cave argues that legacy is the driving force behind works of art, music, literature, science, culture, architecture and other artifacts of civilization. How? Because of something called Terror Management Theory. Awareness of one’s mortality focuses the mind to create and produce to avoid the terror that comes from confronting the mortality paradox that would otherwise, in the words of the theory’s proponents—psychologists Sheldon Solomon, Jeff Greenberg and Tom Pyszczynski—reduce people to “twitching blobs of biological protoplasm completely perfused with anxiety and unable to effectively respond to the demands of their immediate surroundings.”

Maybe, but human behavior is multivariate in causality, and fear of death is only one of many drivers of creativity and productivity. A baser evolutionary driver is sexual selection, in which organisms from bowerbirds to brainy bohemians engage in the creative production of magnificent works with the express purpose of attracting mates—from big blue bowerbird nests to big-brained orchestral music, epic poems, stirring literature and even scientific discoveries. As well argued by evolutionary psychologist Geoffrey Miller in *The Mating Mind* (Anchor, 2001), those that do so most effectively leave behind more offspring and thus pass on their creative genes to future generations. As Hitchens once told me, mastering the pen and the podium means never having to dine or sleep alone.

Given the improbability of the first three immortality narratives, making a difference in the world in the form of a legacy that changes lives for the better is the highest we can climb up Mount Immortality, but on a clear day you can see forever. ■

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SCIENTIFIC AMERICAN MARKETPLACE

My 300 millionth cousin



www.evogeneao.com

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My 175 millionth cousin

Fig. 1 - Product of evolution.

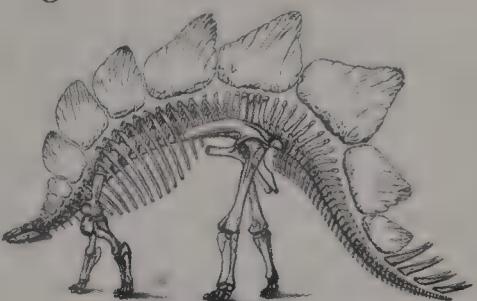


Fig. 2 - Product of intelligent design.



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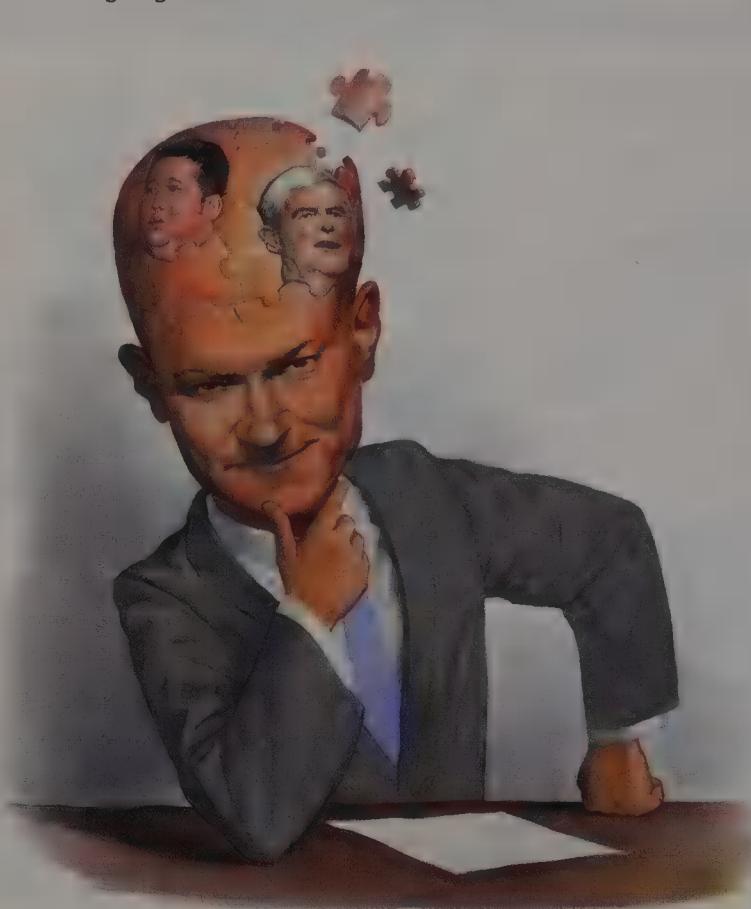


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The Doctor Is Way Out

An amateur examination of one shrink's noodle

This column is not about Newt Gingrich. Nor is it about Chaz Bono. It's not even about how the thought of them dancing together would make Rick Santorum's head explode. No, this column is about a psychiatrist named Keith Ablow, who in recent months has taken the time to write about Gingrich and Bono from his unique perspective as a mental health professional.

According to his Web site, Ablow "serves as the FOX NEWS expert on psychiatry." It was in that capacity that in January, Ablow penned a widely circulated column regarding the well-documented peccadilloes of Gingrich and the former House speaker's qualifications to be president.

"Here's what one interested in making America stronger can reasonably conclude—psychologically—from Mr. Gingrich's behavior during his three marriages," Ablow wrote. "Three women have met Mr. Gingrich and been so moved by his emotional energy and intellect that they decided they wanted to spend the rest of their lives with him." He continued, "Two of these women felt this way even though Mr. Gingrich was already married." He dug deeper, "One of them felt this way even though Mr. Gingrich was already married for the second time, was not exactly her equal in the looks department and had a wife (Marianne)

Steve Mirsky has been writing the Anti Gravity column since a typical tectonic plate was about 33 inches from its current location. He also hosts the *Scientific American* podcast *Science Talk*.



who wanted to make his life without her as painful as possible."

Now buckle up for the exciting psychological conclusion: "When three women want to sign on for life with a man who is now running for president, I worry more about whether we'll be clamoring for a third Gingrich term, not whether we'll want to let him go after one."

I am not a mental health professional, nor do I play one on TV. Nor am I about to claim that Gingrich is a psychopath. But I do wonder whether the doctor's personal political views may be overriding his medical judgment.

For example, Ablow is surely familiar with the Hare Psychopathy Checklist, used for diagnosing that serious personality disorder. If I were a psychiatrist—which again, I am not—who wanted to publicly contend that there was significant evidence for Gingrich being a psychopath—which I certainly do not—I could pretty much just quote from the checklist: glibness, grandiose sense of self-worth, being manipulative, having poor behavior control, being sexually promiscuous, having many short-term marital relationships and, my favorite for the guy still running for president as this column goes to press, lack of realistic long-term goals.

Clearly, the qualities that Ablow thinks recommend Gingrich to the electorate could easily be interpreted quite differently by some other hypothetical qualified psychiatrist. Someone who might come to this *Seinfeld*-ian conclusion, succinctly stated by the disapproving father of a woman who dated George Costanza's dad: "This guy ... this is not my kind of guy."

Which brings us to Chaz Bono, a guy who used to be a gal. When Bono was on *Dancing with the Stars* last fall, Ablow advised parents "to not allow their children to watch the episodes in which Chaz appears." He contended that to see Bono being applauded could kindle gender dysphoria in vulnerable youths.

Have I mentioned that I'm not a psychiatrist? Therefore, I can't say whether Ablow is correct—although it's easy enough to find vehement disagreement on this issue from other psychiatrists online. I recommend the response from Jack Drescher, a member of the DSM-5's Sexual and Gender Identity Disorders Work Group, who describes the Fox News maven's views as having "little basis in current clinical practices" and being basically just "opinions, scare tactics and inflammatory language."

What I can't figure out is why Ablow did not also offer additional obvious advisories. For example, girls who watch *Dancing with the Stars* might manifest a sudden desire for breast augmentation surgery, and boys who watch might exhibit a predilection for wearing toreador pants tight enough to lower sperm counts. My advice: people who sit on the couch to see fox-trots are better off than people who lie on the couch to hear Fox psych. ■

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April 1962

Space Race

"The success of Project Mercury's first manned orbital flight on February 20 may

have set the stage for international co-operation in the exploration of space, as well as demonstrating through the performance of the astronaut John H. Glenn, Jr., that men have a useful function in space vehicles. Glenn demonstrated that he could 'fly' the capsule, controlling its pitch, yaw and roll after malfunctions in the automatic system developed early in the flight. Glenn later said his experience indicated 'that a man can take over control of the various systems.' In fact, he suggested, 'we probably can go on some future flights with considerably less automation and less complexity.'"

Nuclear Arsenals

"It is clear that military arguments alone are not likely to be dominant in U.S. discussion of a possible drastic first step toward nuclear disarmament. This is widely admitted in the U.S., where the impediments to disarmament are being seen more and more as economic, political and emotional in origin rather than as based on operational military considerations. A vital aspect of the problem for the U.S. is the effect that drastic disarmament steps would have not only on the economy as a whole but also on those special sections of high-grade, science-based and highly localized industries that are now so overwhelmingly involved in defense work."

supposedly the safest steamship afloat, while steaming on her proper course, on a clear, starlit night, struck an iceberg and within a few hours sank, carrying down with her over sixteen hundred souls. The technical lessons taught by this prodigious disaster are three: First, that the naval architect has not yet learned how to make an absolutely nonsinkable ship, and that probably he never will. Second, that if every ship is sinkable, it should carry at least a sufficient number of lifeboats to take care of every person on board until other ships, summoned by wireless, can reach the scene of a disaster. Third, that the transatlantic sailing route for passenger steamships should be shifted



SCIENTIFIC AMERICAN, VOL. CVI, NO. 17; APRIL 27, 1912

April 1912

Loss of the *Titanic*

"On Sunday, April 14th, the largest and

The Agenda Setters

Bringing Science to Life

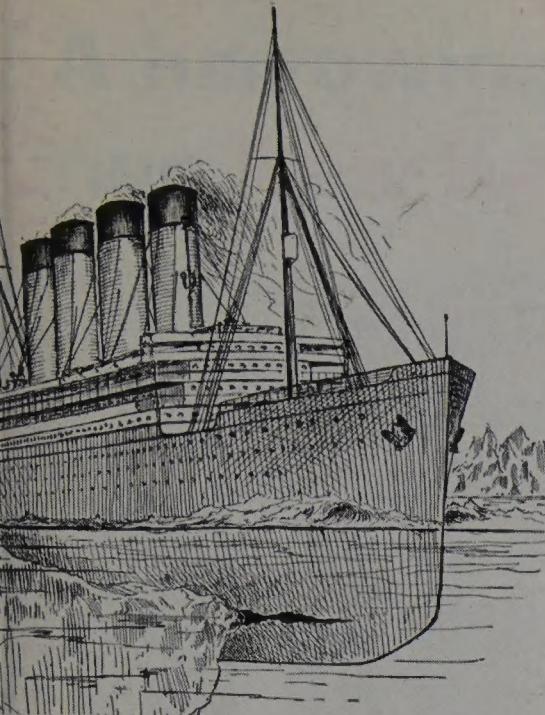
Digital Health Summit

Consumer Electronics Show | Las Vegas | January 11, 2012

Three of the most influential healthcare professionals—Greg Lucier of Life Technologies, Eric J. Topol, M.D. of Scripps and Reed Tuckson, M.D. of UnitedHealth Group—discussed genetics, the future of wireless health, the promise of personalized medicine and managing healthcare costs at SCIENTIFIC AMERICAN's standing-room-only panel at CES. Later that day, thought-leaders gathered at SA's "salon" for cocktails and conversation.



Photos by FRED FISHKIN



Titanic doomed: A glancing collision with an iceberg popped open hull plating on several compartments of the ship (the "gash" was much smaller than depicted here).

so far south as to be entirely beyond the track of floating icebergs."

For a collection of articles from 1912 on the

Titanic disaster, including editorials, an overview of the ship and safety issues, a plan for carrying more lifeboats, and the science of icebergs, see www.ScientificAmerican.com/apr2012/titanic

Blood Doping

"Sir Edwin Ray Lankester has inquired if the Swedish authorities, who will have charge of the coming Olympian games, will permit a Marathon competitor to carry an oxygen tank or bag and take from it an occasional whiff during that cruel and grueling twenty-six odd miles that must be run. 'As oxygen is not a drug, but as natural an article of consumption as water, there seems to be no reason why the runner should be disqualified for refreshing himself with it, as he may with soup or water.' Sir Edwin's proposal is amazingly unscientific in a scientist of so great reputation; and it is most unsportsmanlike."



April 1862

Whiskey vs. Cannon

"In a recent proclamation Governor Brown of Georgia commands the people of that State to cease the manufacture of ardent spirits after the 15th of March, on pain of having their stills seized for the use of the government. The proclamation concludes as follows: 'We need more cannon with which to meet the enemy. Gun-metal used in the manufacture of field pieces is composed of ninety parts of copper and ten of tin. The copper stills of Georgia, which are now heavy columbiads [large-bore cannon] of destruction aimed against our own people, would, if manufactured into cannon, make many a battery of six pounders, to be turned against the enemy.'"

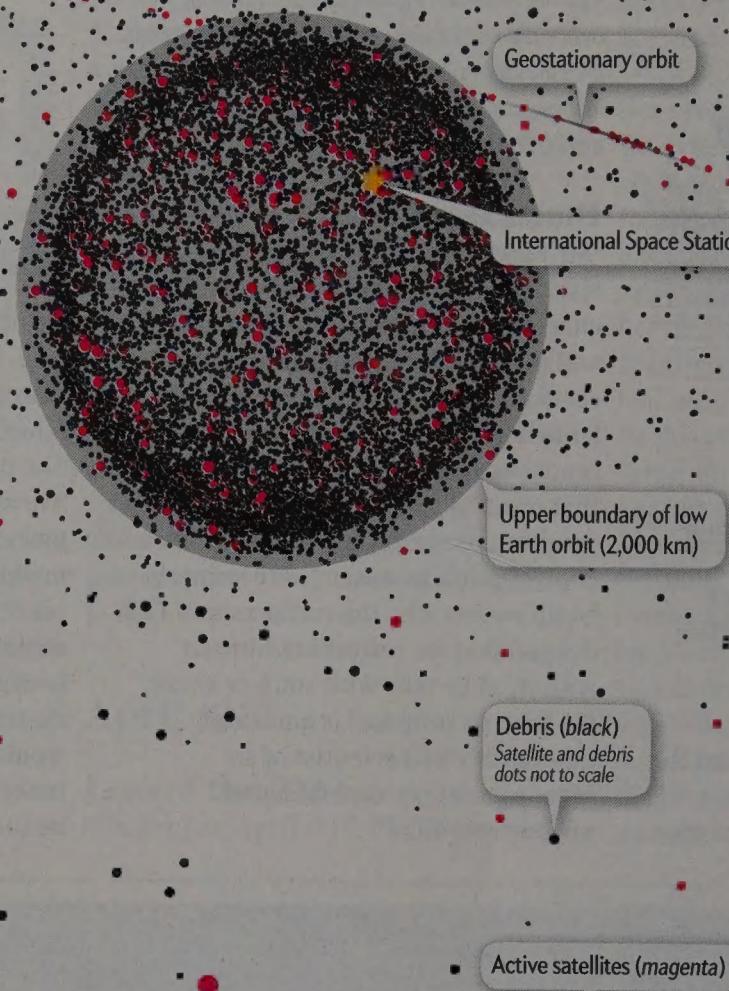


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Space Age Wasteland

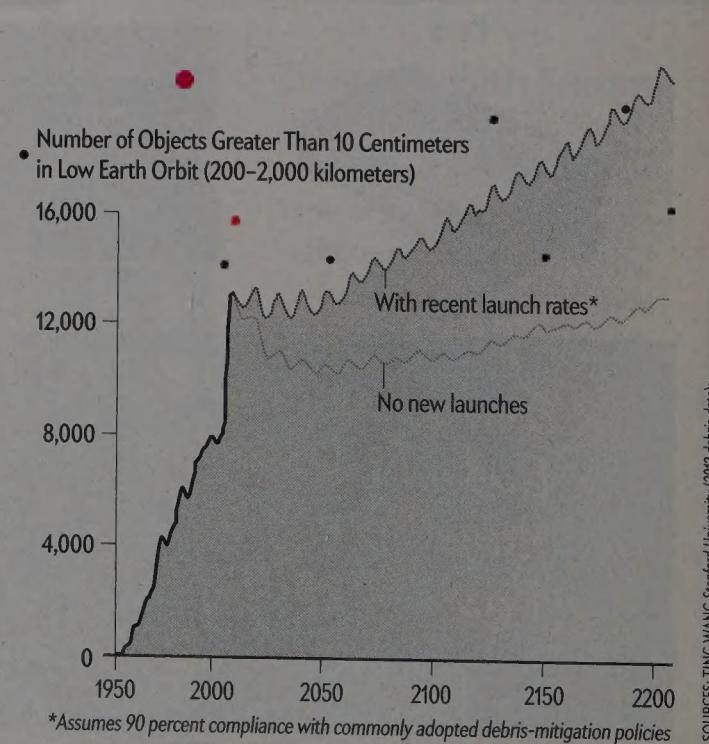
Debris in orbit is here to stay

Space may be incomprehensibly vast, but Earth's environs are crowded with junk. Spent rockets, derelict spacecraft, satellite fragments and loose hardware now form a cloud of debris that poses a threat to orbiting satellites and astronauts. Sky watchers have catalogued more than 16,000 objects larger than about 10 centimeters, most of them in low Earth orbit, at altitudes of 200 to 2,000 kilometers (right).

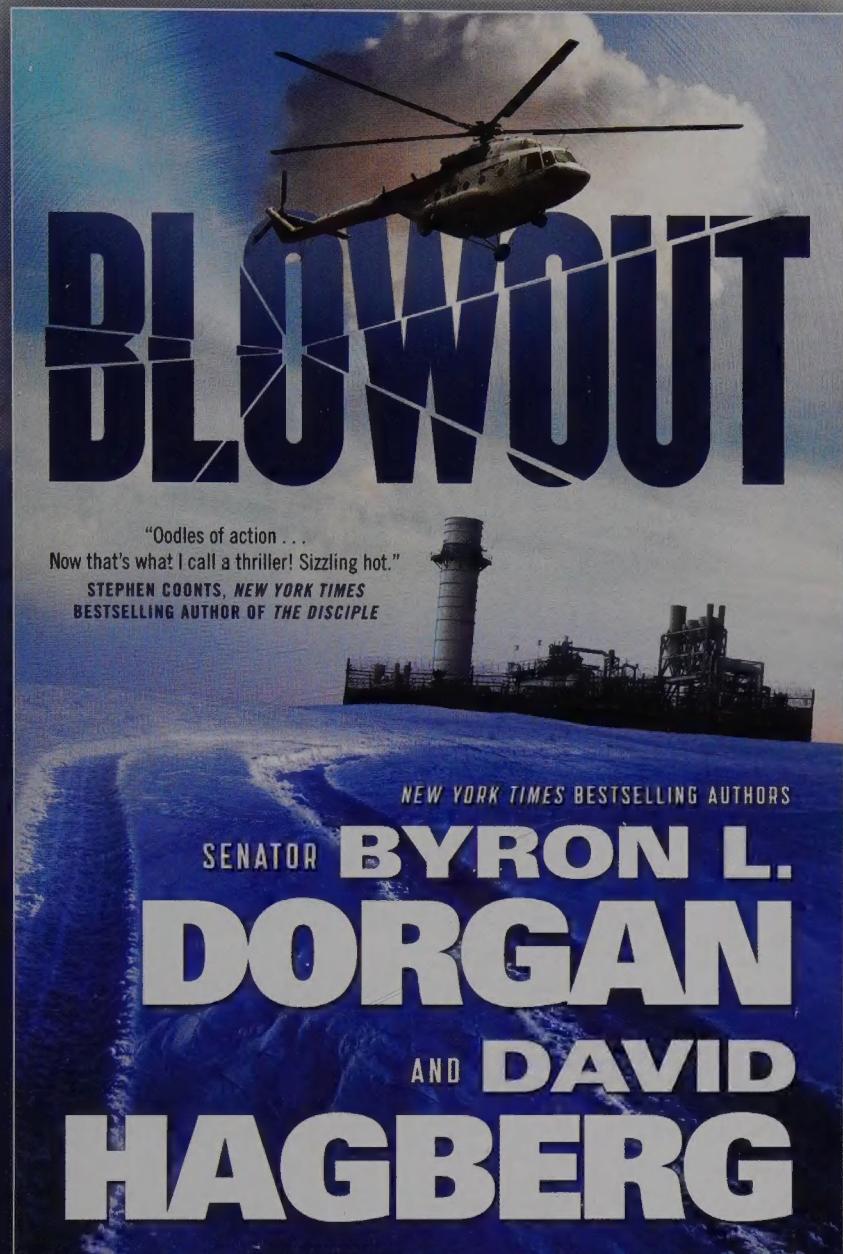
And the junk is self-sustaining. If humankind were to cease all spacefaring activities, the hardware we have already cast off would continue to collide and fragment into bits for centuries. Maintaining current launch rates would make the problem even worse. The number of space objects has shot up in the past five years because of China's 2007 test of an antisatellite weapon and the 2009 crash between Russian and U.S. satellites. Governments are contemplating cleanup measures but have yet to devise a workable solution. —John Matson

SCIENTIFIC AMERICAN ONLINE

See more data in an interactive graphic at ScientificAmerican.com/apr2012/graphic-science



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SENATOR BYRON L. DORGAN served as a US Senator and Congressman for North Dakota for thirty years before retiring in January 2011. He was Chairman of Senate Committees and Subcommittees on the issues of Energy, Aviation, Appropriations, Water Policy and Indian Affairs.

DAVID HAGBERG is a former Air Force cryptographer who has traveled extensively in Europe, the Arctic, and the Caribbean. He has published more than twenty novels of suspense, including the bestselling *Allah's Scorpion*, *Dance with the Dragon*, and *The Expediter*.

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